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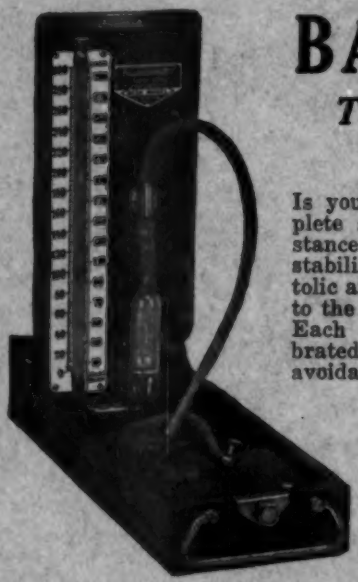

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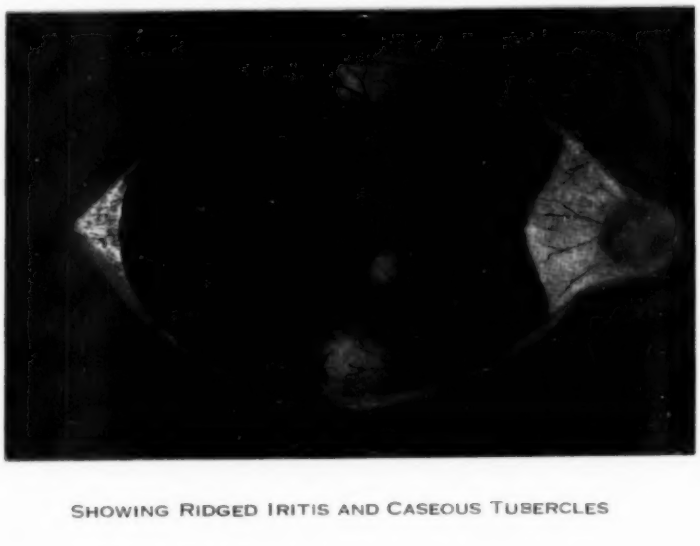
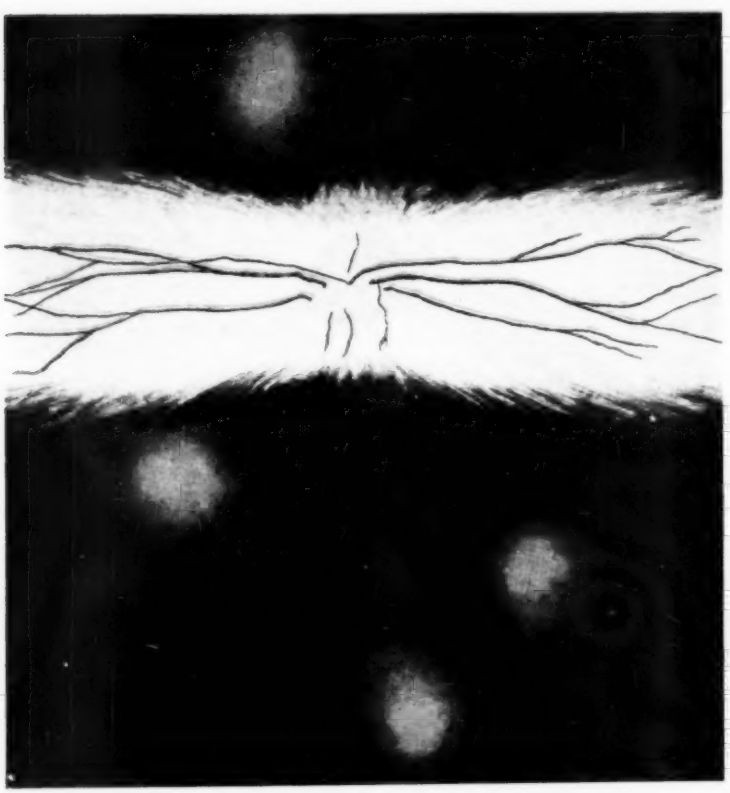


SHOWING RIDGED IRITIS AND CASEOUS TUBERCLES



RABBIT'S FUNDUS, SHOWING EARLY TUBERCLES OF CHOROID

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Vol. 7 FEBRUARY, 1924 No. 2

CHANGES FOUND IN EYES OF RABBITS FOLLOWING INJECTION OF LIVING TUBERCLE BACILLI INTO THE COMMON CAROTID ARTERY.

WILLIAM C. FINNOFF, M.D.

DENVER, COLO.

This paper briefly reviews previous experimental work in this direction. Clump emulsions were used; the average incubation period was a little over six days. Characteristic lesions were studied in the iris, cornea, conjunctiva, lids, episclera, choroid, retina and retinal vessels and in an extraocular muscle.

Friedrich and Nösske[1] were evidently the first investigators to discover tuberculosis occurring in the eyes of animals which had been inoculated with tubercle bacilli. A report of their work was published in 1899. They studied the changes that occurred in the body of animals following the introduction of living bacilli of high virulence into the arterial circulation. Rabbits were used and the material was injected into the left ventricle of the heart; tuberculosis of the iris and other organs of the body developed. Their attention was centered on general tuberculosis and not on the ocular changes. They only used the eye as an index to the degree of infection and noted that the presence of iritis indicated a severe generalized disease.

Stock,[2] in 1903, was the first person to describe in detail the symptoms and pathology of experimental tuberculosis of the eye in animals. In the course of his experiments to determine the etiology of sympathetic ophthalmia and endogenous uveitis, he injected tubercle bacilli into the auricular veins of rabbits and found that, in a number of animals, a characteristic iritis and choroiditis occurred. In his series the first manifestations were observed between thirteen and fifteen days after the inoculation. In some animals the lesions healed in two or three months and left scars in the iris and choroid. His experiments proved conclusively that some types of uveitis are due to tuberculosis. Since Stock's work was published, other investigators have studied this subject.

In a paper read before this Society at its last meeting, I described a technic for producing experimental tuberculosis, and in the present essay the results which were obtained after following this method are described.

Emulsions consisting of clumps of living tubercle bacilli were injected into the common carotid artery of rabbits, and lesions in the eyes developed in all of the animals which were inoculated. All types of bovine and human bacilli were used, and the quantity injected was varied. The lesions naturally occurred sooner and with greater severity in the rabbits which were infected with large doses of virulent organisms. When avirulent bacilli were used, the incubation period was longer, and the ocular disease was less severe. Bovine bacilli are more pathogenic for rabbits than human bacilli, and they produce a more violent form of ocular tuberculosis. The quantity of bacilli (in the emulsions) which were used in the experiments varied from 0.2 to 10 mg. Ocular lesions occurred on the side injected, without exception, in the 46 animals which were used in this series. Only two of the rabbits showed evidence of disturbance of the brain. The majority of them developed pulmonary, hepatic, and a more or less generalized tuberculosis. They usually died before the eye condition had run its full course. Out of the whole series, three animals recovered and showed healed lesions in the eye. This contrasts strikingly with the results which we have obtained after the injection of dead bacilli. In the latter experiments the animals usually lived, and the ocular disease ran its full course.

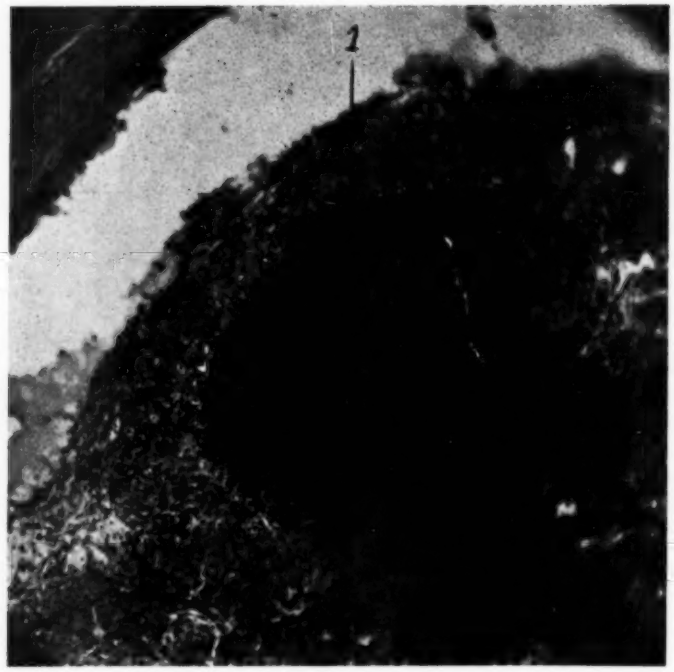

Fig. 1. Area of caseation in iris.

A paper covering this phase of the investigation will be read in the near future.

In this series in which living bacilli were used, the average incubation period of ocular tuberculosis was a little over six days. This is a much shorter period than was reported by Stock in his series of experiments. The closer proximity of the carotid artery to the eye and the use of a clumped emulsion account for this difference. Out of the 46 animals that were used, the incubation period was as follows: First twenty-four hours, 3 animals; second day, 4; third day, 1; fourth day, 12; fifth day, 2; sixth day, 4; seventh day, 7; eighth day, 4; ninth and tenth days, none; eleventh day, 7; twelfth

Fig. 2. Opacification of the cornea with a ring of newly formed blood vessels at the limbus. Early conjunctivitis with a small quantity of secretion on the margin of the lower lid.

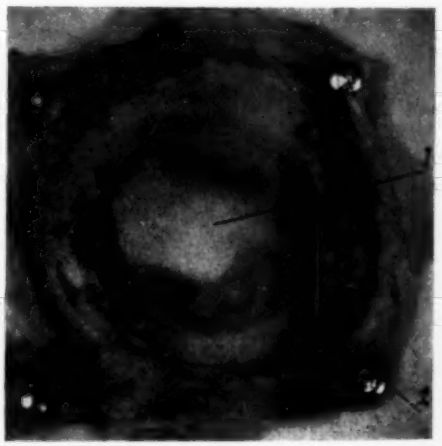

Fig. 3. Enucleated eye. 1, center of cornea opaque. 2, outer darker area due to dense infiltration of substantia propria with fine blood vessels.

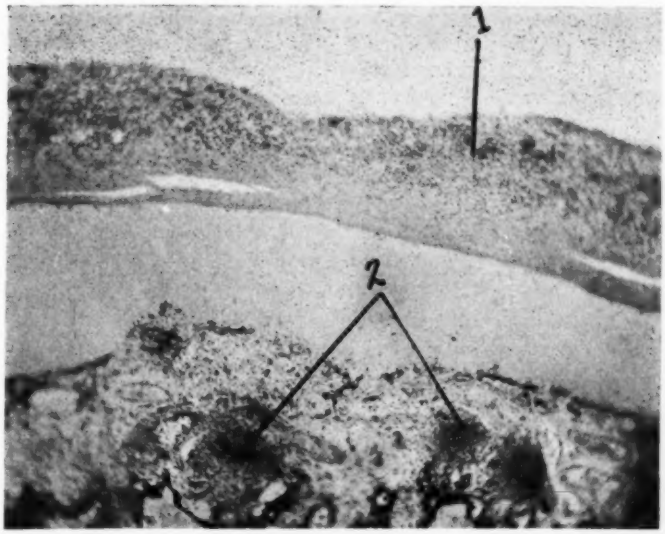

Fig. 4. Loss of corneal substance due to necrossis of anterior layers in its center. 1, New formed blood vessels and cellular infiltration in substantia propria of cornea. 2, Beginning tubercles of iris.

day, 1; thirteenth day, none; fourteenth day, 1.

The ocular changes are described in the order of their occurrence. In the majority of animals the first change was a contraction of the pupil in the eye corresponding to the side which had been injected. It was frequently seen several days before the appearance of other manifestations. Contraction of the pupil occurs after other irritating substances have been injected into the carotid artery, and the essayist is inclined to believe that this is the result of either mechanical or chemical irritation and is not specific to tuberculosis.

The first striking changes were usually seen in the iris. They varied with the severity of the attack. In the more acute form, hemorrhages occurred into the iris and anterior chamber, and a turbid aqueous was occasionally seen on the day following the inoculation. This phenomenon has also been observed after the introduction of other irritating substances into the arterial circulation and was probably the reaction to an irritant and not specific.

As a rule, characteristic symptoms of tuberculosis were not seen until after the fourth day, when the iris became irregularly thickened in the form of pronounced ridges, which were triangular in shape, with their base directed toward the ciliary portion of the iris and the apex toward the pupil. To this form, which occurred in most cases, I have given the name of ridged iritis (Plate 1). In the severe cases, very fine blood vessels appeared on the surface of the ridges. Serous exudates were also seen on the lens capsule at the pupillary border of

Fig. 5. A, Ulcer of bulbar conjunctiva. 1, Opacity in corneal center. 2, New formed blood vessels in cornea.

the iris, in the region of ridges. In the less severe cases an avascular ridged iritis was the only change that occurred in this portion of the eye. It was seen in the three animals which recovered, and the terminal result was a uniform atrophy of the iris stroma. In the remainder of the animals, the condition was progressive, and a few days after the onset of ridged iritis, grayish-yellow, round and oval areas were seen pushing thru the iris stroma. They were usually located in the base of ridges at the upper and lower pole of the iris. Occasionally, one developed near the pupil. The nodules increased in size rapidly and finally pushed thru the iris stroma and protruded into the anterior chamber. New formed blood vessels ran over the surface of the majority of the nodules (Plate 1). In some cases these masses filled the angle

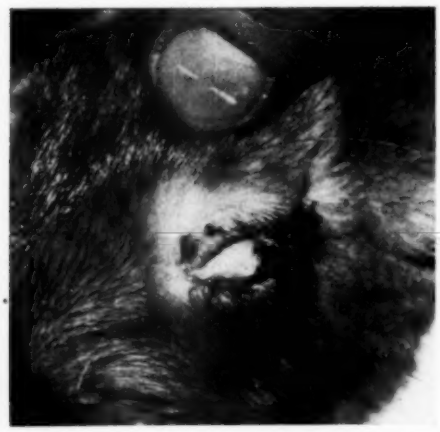

Fig. 6. Characteristic conjunctival secretion. Mild case.

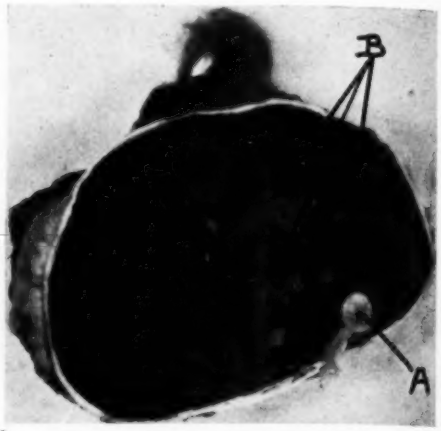

Fig. 7. Posterior portion of eye from which the retina has been removed. A, Atropic patch in choroid with tag of retina adhering to scar. B, Scattered patches of disturbed pigment.

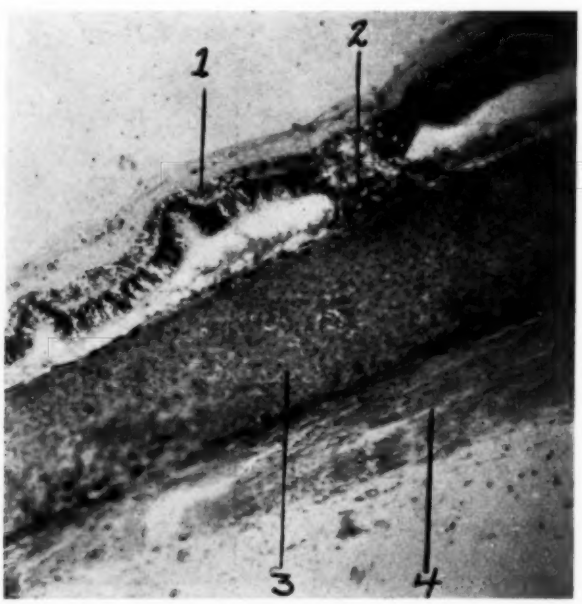

Fig. 8. Showing early stage of tuberculosis of choroid. 1, Retina. 2, Proliferated pigmented epithelial cells. 3, Epithelioid cells. 4, Sclera.

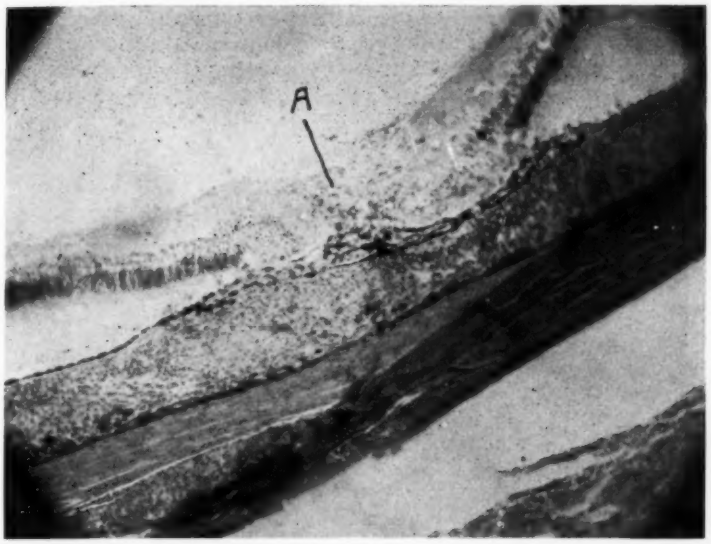

Fig. 9. Showing choroidal tubercle, proliferation of pigment epithelium and degeneration of retina. A, Complete absence of retinal elements.

of the anterior chamber and were associated with opacification of the adjacent cornea. Microscopically, the nodules were found to be caseated tubercles in the iris (Fig. 1). In most of the animals the nodules gradually increased in size and remained vascular. In some of the milder cases they developed to a certain point, and with the exception of the disappearance of blood vessels on the surface of the mass, they remained stationary during the remainder of the animal's life. All of the animals in which caseous tubercles in the iris occurred died of generalized tuberculosis or other complications before healing had taken place, and no further changes were seen in the iris.

In the fulminating cases a general stippled haziness of the cornea appeared a few days after the onset of the ridged iritis. This progressed rapidly, and in two or three days the whole cornea became white and resembled coagulated albumin (Figs. 2 and 5). A dense network of blood vessels soon appeared in the whole circumference of the cornea at the limbus. They continued to invade the substantia propria from the periphery toward the center, and in about ten days' time the whole cornea became vascular. The vascularity of the cornea resembled the socalled salmon patch of human interstitial keratitis (Fig. 3). Sections of the eye, in these cases, show necrosis of the center of the cornea with new formed blood vessels, leuco-

Fig. 10. Caseous area in choroid with thinning of pigment epithelium over center of tubercle. Atrophy of retina over caseous area. 1, Sclera. 2, Caseated area in choroid. 3, Atrophied choroid.

cytes, and fibroblasts in the substantia propria toward the periphery (Fig. 4). This type of corneal involvement is not confined to tuberculosis. It occurs after the injection of other substances which alters the nutrition of the cornea. Corneal changes simulating human corneal tuberculosis did not occur in animals ulcers of the bulbar conjunctiva near the limbus occured (Fig. 5). Later, the secretion became thicker and more profuse. The secretion did not resemble pus. It was thicker and white in color, resembling in appearance and consistency casein in sour milk (Fig. 6). In 9 out of 20 smears

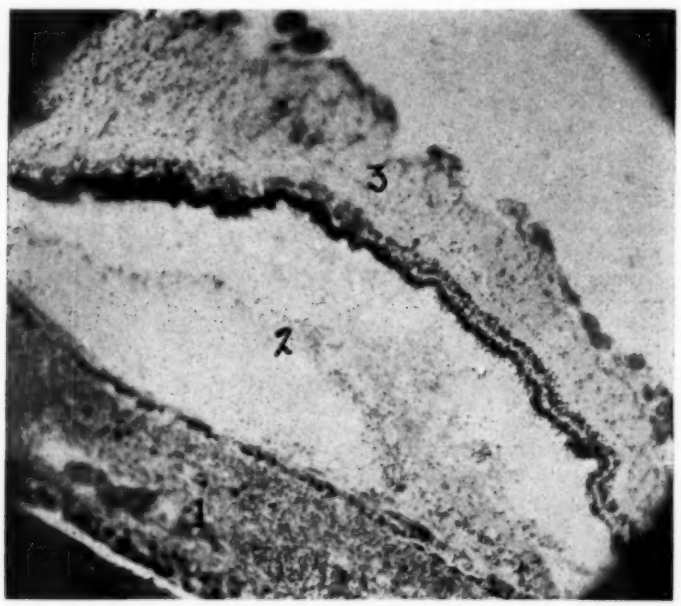

Fig. 11. Showing detachment of retina by a serocellular exudate in front of choroidal tubercle. 1, Choroidal tubercle. 2, Serous exudate. 3, Retina.

any of the animals which had been inoculated with living tubercle bacilli. However, changes of this nature have been observed in animals in which dead bacilli were used. The findings in these cases will be described in a subsequent paper.

Conjunctivitis occurred in all of the animals that had corneal tuberculosis. It was usually a later manifestation than the lesions which have just been described. In the severe cases it began at the time that the corneal opacities appeared. In the milder cases it did not appear until after the fourteenth day. In the beginning the conjunctiva was severely congested and thickened, and a small quantity of secretion was seen. Red and yellow nodules then appeared in the conjunctiva. In some of the animals these nodules resembled phlyctenules. In several instances the elevations broke down and formed small irregular ulcers. In two tubercle bacilli were found in the secretion.

Ulcers of the lid occurred in four animals. They were first observed at the time of the onset of the conjunctivitis. They were located at the lid margin, and in the beginning were seen as small round elevations which soon broke down and formed ulcers. The ulcers were irregular excavations with mouse-eaten edges. The surface was covered with dirty gray necrotic material, thru which small red points of granulation tissue protruded. They were chronic in nature, and none healed before the animals died.

Episcleritis was also a late manifestation. It occurred in three animals and appeared after the fourteenth day.

Choroiditis was not seen ophthalmoscopically until after the fourteenth day. It was observed in all the animals in which opacities of the media did not interfere with the ophthalmoscopic ex-

amination. Ill defined oval patches, which were lighter in color than the surrounding fundus, were the first changes which occurred. These soon became yellow in color, without any disturbance of the pigment (Plate II). From ten days to two weeks after the development of the yellow patches, pigment granules began to appear on the surface of the masses, and the tubercles became clearly circumscribed. The changes from this point were very gradual, and in the three animals which survived it was possible to follow the steps from active inflammation to atrophy. The pigment which was seen on the surface of the tubercle gradually moved toward the periphery of the lesion and was deposited there. In the atrophic stage the lesions were seen as brilliant white, round and oval areas, with a few pigment granules scattered over the surface, and pigment banked up at the margin of the lesion, or as irregular peppered patches of pigment.

Histologically, the sequence of events in the background of the eye were: first, an infiltration of the choroid with epithelioid cells (Fig. 8). In some sections clumps of bacilli were found in the center of the lesion. In the early stage the pigment epithelium was not disturbed. This corresponded to the first change that was seen in the living eye with the ophthalmoscope (Plate II). Later, small round cells surrounded the collection of epithelioid cells and the pigment epithelium then showed degeneration and proliferation. The retina then became disintegrated and lost its normal appearance. The pigment cells and pigment granules migrated forward (Fig. 9). This corresponded to the second stage of the ophthalmoscopic picture. The next stage was caseation in the center of the lesion, with complete atrophy of the retina over the caseous area and disappearance of some of the pigment in the center.

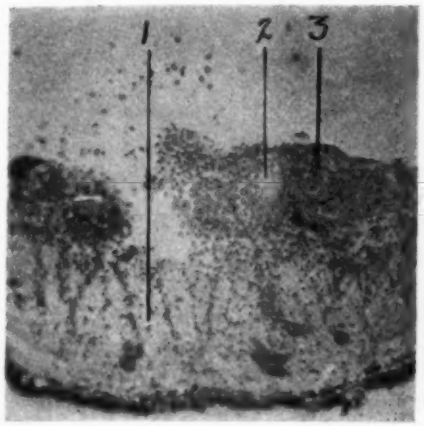

Fig. 12. Generalized uveitis with dense white exudate in vitreous.

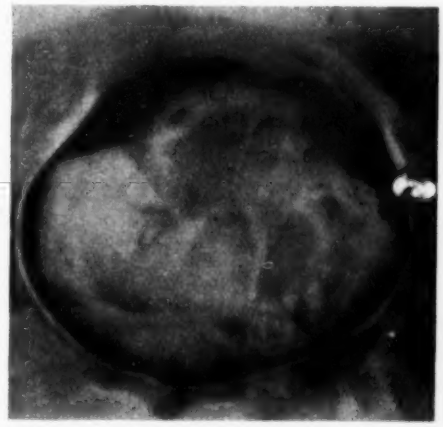

Fig. 13. Showing perivasculitis of retinal vessels. 1, Nerve fiber layer of retina. 2, Retinal vessel. 3, Epithelioid cells.

Often localized detachments of the retina occurred over choroidal tubercles. These were seen as brilliant, pearl like areas in the fundus. They gradually increased in size in the severe cases. The detachments were due to a serocellular exudate which separated the retina from the infiltrated choroid (Fig. 11).

A generalized severe uveitis occurred in the majority of the animals. The vitreous became cloudy and obscured the fundus for ophthalmoscopic examination. In sections of these eyes a severe generalized uveitis, with marked thickening of the choroid, ciliary body, and iris, was always found (Fig. 12).

Tuberculosis of the retinal vessels was observed in only one of the 46 animals. It was seen ophthalmoscopically as a gray, perivascular exudate surrounding the retinal vessels. Choroidal and iris tuberculosis were also

present. The animal had lost weight and was killed on the sixteenth day after the inoculation, so that the specimen might be obtained before the animal died. Microscopic examination of sections of the eye revealed the presence of a marked perivasculitis (Fig. 13). In one of the veins an endothelial thrombus was in its formation, and the vein was half obstructed by newly proliferated endothelial cells from the inner wall of the vein.

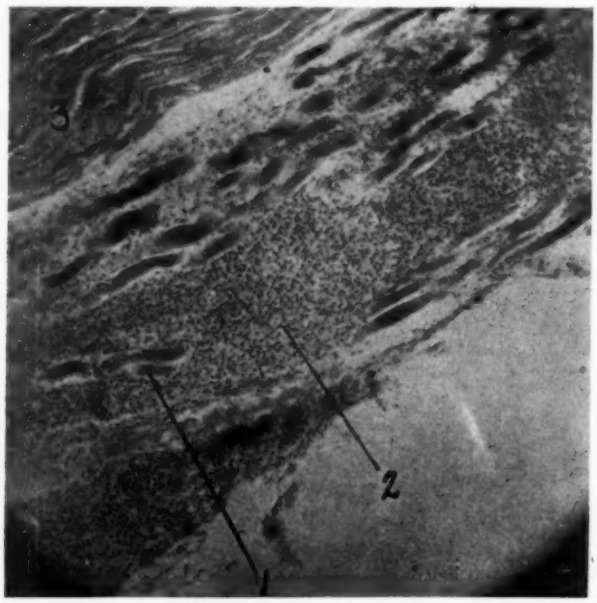

Fig 14. Infiltration of extraocular eye muscle with epithelioid cells. 1, Muscle fiber. 2, Epithelioid cells. 3, Sclera.

A tubercle was found in an extraocular muscle in one of the sections (Fig. 14). Sections also revealed extension of tuberculosis from the interior of the eye thru the sclera into the extraocular structures.

This series of experiments confirmed Stock's conclusions and proved that tuberculosis of hematogenous origin attacks all the structures of the eye excepting the lens.

The practical conclusions that are drawn from these experiments are summarized as follows:

Hematogenous eye infections with living tubercle bacilli are of a severe and progressive type, and with few exceptions do not correspond to the types of ocular tuberculosis which occur in the human being.

In the exceptional, milder cases, tubercles of the iris and choroid run a course which is similar to that seen in the human being.

The first objective symptoms are seen in the iris, next in the cornea, then the conjunctiva and lids, and then the choroid.

The typical iris findings are ridged iritis and caseous tubercles.

The tubercles of the iris usually have blood vessels running over their surface.

Corneal changes that are typical of tuberculosis do not occur frequently after the injection of living tubercle bacilli.

The results that have been obtained in these and later experiments emphasize the well known clinical fact that tuberculosis of the eye, which occurs in subjects who have an active tuberculosis elsewhere in the body, is usually severe and progressive and frequently necessitates enucleation of the eyeball, while the chronic types occur in persons who have either a mild or latent tuberculosis.

For a practical experimental study of the nature of tuberculosis of the eye as it usually occurs in the human being, it is necessary to use small doses of

avirulent living or emulsions of attenuated or dead tubercle bacilli.

The author wishes to express his appreciation for the help and cooperation which he has received from Dr. H. J. Corper, and to the Research Department of the National Jewish Hospital for Consumptives in Denver, which institution has made it possible to carry out these experiments.

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XEROPHTHALMIA, KERATOMALACIA AND XEROSIS CONJUNCTIVAE.

OLAF BLEGVAD, M.D.

COPENHAGEN, DENMARK.

Xerophthalmia is here considered as a deficiency disease with especial reference to its occurrence in Denmark in 1909-1920. The literature of keratomalacia and conjunctival xerosis is reviewed; also the experimental work that has established the importance of fat-soluble vitamin A. The disease may be caused by deficiency thru food lacking this vitamin or because of deficient absorption due to digestive disease, increased consumption of the vitamin during rapid growth or severe disease. Deficiency of the vitamin in the food of the mother during gestation or nursing or in the food of cows depended upon for milk, the use of skimmed milk or of vegetable fats as a substitute for butter also produce it. The responsibility of all these is traced. The mortality and blindness caused are discussed. The treatment includes a corrected diet including large amounts of vitamin. The material on which this paper is based included 434 cases of keratomalacia in children and 13 in adults.

INTRODUCTION.

Xerophthalmia was, in older times, a rather infrequent disease in Denmark, as in most of the European countries; but during the years 1909-1920, and more particularly in the first part of this period, a striking number of cases of this disease occurred in this country. The increased frequency of the disease proved to be special for Denmark and was not found in our neighboring countries. It might, therefore, be supposed to originate in some circumstances special for Denmark, presumably in the economic-nutritional conditions. Presuming, therefore, that an examination of these conditions and the appearance of the disease in this country might throw some light on the genesis of the xerophthalmia, I have gathered all of the cases of keratomalacia observed by Danish oculists—in all 434 cases of keratomalacia in children and 19 cases in adults—and also 148 cases of xerosis of the conjunctiva without keratomalacia, all of which were observed during the years 1909 to 1920.

SHORT HISTORIC SUMMARY.

Xerophthalmia has been known since the middle of the last century. *Johannes Nepomuk Fischer* mentions in his treatise on ophthalmology, published in 1846, a particular necrosis of the cornea which, from the description, must be keratomalacia; and in the treatise of F. Arlt[1], published in 1851, we find 2 cases of "Malacie der Hornhaut" which undoubtedly must have been keratomalacia. The first minute description of the disease, however, was given by Albrecht v. Graefe[8] in 1866. The disease is here called "Hornhautverschwärung," and was by v. Graefe connected with the encephalitis infantilis, described shortly before by Virchow. The pathologic-anatomic alterations found by Virchow in this disease proved, however, afterwards to be normal conditions in the brains of babies. The same year Gama Lobo[7] described the keratomalacia in negro children in Brazil. The origin of the disease, which Gama Lobo called "ophthalmia brasiliana" was thought to be cellular atrophy on account of bad nutrition. The first symptom of xerophthalmia, the xerosis

conjunctiva, was first described by Bitôt[3], in 1863. This description was based on 29 cases of xerosis conjunctivae which appeared together with hemeralopia in children of a larger growth in a foundling hospital in Bordeaux. In Petrograd Blessig[6] observed in the year 1866 numerous cases of xerosis conjunctivae in connection with hemeralopia and also, in more severe cases, keratomalacia. The disease appeared principally during the long Lent Quadragesima and was due, in Blessig's opinion, to the derangement of nutrition caused by the fasting. The origin of the disease was, for a time, erroneously considered as an infection with the xerosis bacillus described in 1880 by Colomiatti (Leber[24]), Kuschbert and Neisser[26] Schleich[27], Schulz[28], but later investigators are now of the opinion that it is mostly due to nutritional phenomena. Thus R. Teuscher[9] and de Gouvea[23] mention in 1867 and 1883 keratomalacia in negro children from the coffee plantation of Rio de Janeiro, where nutritional conditions were said to be very bad. In 1883 Thalberg[25] mentioned keratomalacia in nurslings whose mothers, on account of the Russian Lent Quadragesima, were in very bad nutritional conditions, as their food consisted of rye bread, potatoes, porridge, cabbage, mushrooms and onions. Another doctor in Petrograd. Kubli[31] observed in 1887 a number of cases of this disease, likewise during the Russian Lent Quadragesima. The same year a. case of keratomalacia is mentioned by Weeks[33] of New York, who is also of the opinion, that it is due to bad nutrition. Baer[68], in 1901, observed a case of keratomalacia in a child nourished upon oatmeal gruel. It was restored to health by treating with cow's milk and limewater. The year after Hamburger[75] described a similar case, where the child was cured by the nurse's milk.

Here in Denmark the first case of keratomalacia was described by Gordon Norrie[44] who observed the disease in a child of 8 months, who during 6 weeks had lived upon gruel. In 1903 the Danish oculist Edmund Jensen[76] mentions 9 cases of xerophthalmia which appeared after nutrition with Mellin's food, barley broth and the like. In Japan Mori[82] observed in the years before 1904 numerous cases of xerophthalmia (called "hikan") in children from 2 to 5 years of age. Mori and later on Ishihara[116], are of the opinion that the disease is due to lack of fat stuff in the food. These children lived upon rice, farinaceous food and vegetables. After having been nourished with the mother's milk until one year of age, the Japanese children don't get any milk. Butter, cheese and milk are scarcely known as ordinary food in Japan, as breeding of cattle is very little developed. On the coast, where good fish is to be had, "hikan" was seldom observed. In 1907 Schiele[92] mentions the keratomalacia, of which he has seen many cases in Kursk during the Lent Quadragesima, and he thinks they are due to the lack of fats. Cod liver oil has a special curative value. The cases observed by Schiele appeared partly in children who during the Lent did not get any milk at all, partly in nursing children whose mothers suffered from hemeralopia, even if they had sufficient milk in their breasts. Schiele makes the interesting observation that the nursing children are restored to health without any treatment, if only the mother takes cod liver oil. Flemming[95] mentions 28 cases from Breslau, and Stephenson[101] 31 cases of keratomalacia from London. Both consider the bad nutrition as the cause. Keratomalacia has, in a number of cases, been observed in children affected with "Mehlnährschaden". Pieper[90], Romahn[105], Peters[111], Brünnig[111], Gille[113], Kapuscinski[108]. Rönne[137], in 1916, maintains that the extravagant use of carbohydrates does not play the most important rôle, but that the lack of milk, or "of some substance found in the milk" is the real cause of the disease. Rönne therefore recommends raw milk as a treatment.

That there appears besides the disease of the eye a typical general disease was first admitted by C. E. Block[138, 149]. A minute description of this general disease was published in The Journal of Hygiene," 1921, vol. 19, page 285, to which the reader may be referred. Bloch here points out, on the basis of modern nutritional experiments and on his own observations, the

importance which the vitamin fat-soluble A has to the disease. The same observation is made in a later treatise by R. E. Wright[109], who has seen numerous cases of the disease in South India, and by K. Stolte[198] who observes that the disease disappeared in Germany during the war; and Stolte thinks the reason was, that the mothers during the war were premiated for nursing their children themselves.

Many authors have observed that keratomalacia is particularly frequent with disease of the digestive organs, but xerophthalmia has also been observed with other severe diseases. Fischer[1], Bezold[17], Beyer and Trantas[176], have seen keratomalacia with measles, Arlt[1] and v. Graefe[8] with scarlatina, v. Graefe with typhus, Herbert[58] and Foerster[29] with dysentery, v. Graefe with cholera, Spierer[42] with tuberculous meningitis, Cohn[10] and Stephenson[101] with pulmonary tuberculosis, Ringier[45] with influenza, Adler[18] with variola, and Foerster[20] even says that keratomalacia has been observed in connection with puerperal fever. Finally keratomalacia has not infrequently been observed with icterus, Baas[47], Thompson[49], Hori[50], Elschnig[62], Purtscher[66], Groenouw[69], Spassky[91].

XEROPHTHALMIA DEVELOPED BY EXPERIMENTS MADE UPON ANIMALS.

Experiments caried out upon animals during late years have succeeded in developing xerophthalmia, whereby light has been thrown upon its genesis. Already in the year 1909, P. Knapp[96] succeeded in developing a disease of the eye in rats when they were fed with egg albumen, starch, glucose, pork grease, water and salts. Later McCollum, Osborne and Mendel, Hopkins and others observed the disease of the eyes in rats fed with food lacking certain fats. But that the rats' disease was identical with the keratomalacia, was not known until 1915. M. Goldschmidt[126] proved that the rat's keratomalacia both clinically and pathologico-anatomically is so much like the human keratomalacia, that we may consider the rats' disease of the eye as an experimental keratomalacia in rats. This was confirmed by later investigations carried out by Emmet[175], Stephenson and Clark[185], Wason[193] and Ejler Holm[194].

Thru the well known elaborate experiments upon animals carried out by Hopkins, Osborne and Mendel, McCollum, Funk and others, we have learned about the presence of the vitamins, fat-soluble A, water-soluble B and antiscorbutic C. Soon afterwards it was discovered that the xerophthalmia appeared in rats, the food of which lacked fat-soluble A, and investigations carried out by Stephenson and Clark[185], Osborne and Mendel[191] and Emmet[175] have proved that the xerophthalmia in rats is due to a diet deficient in fat-soluble A. Also in other animals, for instance, in rabbits Nelson and Lamb[184], in mice A. Auer[161], in dogs Steenbock, Nelson and Hart[192], in guinea-pigs Boock and Trevan, and ducks Rumbaur[197], we have succeeded in developing xerophthalmia.

The disease of the eye in the animals is undoubtedly due to the lack of fat-soluble A in the foodstuffs. It is, therefore, an all but obvious conclusion that the human xerophthalmia should equally be due to the lack of fat-soluble A.

THE VITAMIN: FAT-SOLUBLE A. The primary sources of fat-soluble A are found in the green leaves of plants. Animals are not able themselves to form the fat-soluble A, and what may be found of this stuff in fats and oils derived from the animal kingdom, must therefore originate from the food and primarily from the green leaves of plants. But it is not found in the vegetable oils, tubers and the like that constitute the reserve nutrition for the plants. Abundant fat-soluble A is found in the first food for young mammiferous animals and birds: milk and eggs. There are symptoms that seem to indicate that a human being in contrast to the herbivorous animals is not able to assimilate anything much of the fat-soluble A derived from the plants. The literature mentions xerophthalmia with vegetarians, Kubli[31], Uhthoff[29], Wicherkiewicz[102], Flemming[95]. And among the Japanese children who lived upon rice, farinaceous food and vegetables, Mori[82] counted no less than 1,511 cases of xerophthalmia, among which were 116 cases of keratomalacia.

The animals cannot themselves form the fat-soluble A; they derive it from the food, but the animal organism contains normally a reserve supply. Even if the food does not contain any, the animal may live and grow for a while upon the reserve supply of fat-soluble A, and it is not until this reserve has been exhausted that the growth will cease. The animal then sickens, is seized with xerophthalmia and infections of various kinds (especially pulmonary affections) and finally dies. There is reason to believe that the fat-soluble A is stored up in the body together with the spare fat. The living organism is constantly consuming fat-soluble A, and the consumption is greatest during growth. A young rat will only for a short time do without fat-soluble A in its food, before it dies, the reserve supply being rapidly exhausted. In the grown-up animal, on the contrary, the consumption of fat-soluble A is not so great; it may live a longer time upon the reserve supply.

For the gravid or nursing mother this reserve supply is of a very special importance, inasmuch as she therewith is able to secure to the young ones ample fat-soluble A, even if her own diet for a shorter time should be deficient in fat-soluble A. But if the mother's food for a longer time is destitute of fat-soluble A, her own reserve supply will be exhausted, which will be pernicious to the offspring. The content of fat-soluble A of the mother's milk varies with the content of fat-soluble A of the mother's food. If the mother's food does not contain any fat-soluble A, the mother as well as the suckling is likely to be seized with xerophthalmia, as proved, for instance, by Drummond[152] and by Nelson, Lamb and Heller[196].

The content of fat-soluble A of the cow's milk varies also with the content of fat-soluble A of the cow's food. Thru experiments with cows stall fed during the winter and cows fed upon grass, Steenbock, Boutwell and Kent[160], Drummond, Coward and Watson[187], Hughes, Fitsch and Cave[188], Kennedy and Dutcher[195] have shown that milk from cows stall fed contains considerably less fat soluble A than the milk from cows fed upon pasture. It is, therefore, important for the quantity of the reserve supply with which the young ones are born, how great the content of fat-soluble A of the mother's food has been during the gestation. And also for the supply of fat-soluble A to the sucking young ones, it is of great importance how great the content of fat-soluble A of the mother's food has been in the period when the young ones are living upon the mother's milk.

The fat-soluble A may be destroyed by heating under exposure to the atmosphere (oxidizing), hardening thru hydrogen and being exposed to ozone, or ultraviolet light.

Also other substances than the fat-soluble A are important for originating the xerophthalmia, since this disease is developed more easily in rats when also other parts of the most important constituents of the food, for instance, the quantity of salts or of albumin, are deficient. Too abundant quantities of carbohydrates in the food may probably also be noxious, as they will impede the mobilization of the fat-soluble A in the reserve grease, Hopkins[189].

Is human xerophthalmia a deficiency disease due to the lack of fat-soluble A?

The numerous experiments made upon young rats have established that the rats' xerophthalmia is due primarily to the lack of fat-soluble A. As far as the human xerophthalmia is concerned, there are (as shown by Bloch) so many factors that enter largely into the question, that the matter gets much more complicated than the experiments upon the rats. Still, all the literature concerning the xerophthalmia in human beings and in animals argues that the primary cause of the human xerophthalmia is undoubtedly also the lack of fat-soluble A.

The deficiency of fat-soluble A in the organism may be produced in the following ways:

1. By reduced doses of fat-soluble A.
   - A. Food poor in fat-soluble A.
   - B. Deficient absorption of fat-soluble A in the intestine (digestive diseases).

2. By increased consumption of fat-soluble A.

A. During a rapid growth.

B. During a severe disease.

1. A. In the great majority of cases of keratomalacia we will find that the diet for a longer period has been deficient in fat-soluble A. The literature presents numerous examples of the fact, that the food poor in fat-soluble A has been the sole cause of xerophthalmia—especially in the lighter cases (xerosis conjunctivae). Keratomalacia has even been observed in adults, otherwise quite healthy, who for a longer period have lived upon a diet deficient in fat-soluble A.

1. B. The literature affords numerous examples of keratomalacia in connection with digestive diseases. There are some reasons to believe, that in cases of digestive diseases, the fat-soluble A is not so completely absorbed as when the intestine acts normally; in which case a diet relatively rich in fat-soluble A will act as if it were poor in fat-soluble A. Keratomalacia has been observed in a good many cases in adults affected with icterus, see for instance Hori[150]. It may be presumed, that the poor fat resorption caused by the icterus has caused the deficient resorption of the fat-soluble A, connected with the fats. The fat-soluble A of the human food is nearly always found in connection with fats (butter, cream, yolk of eggs). Of special interest is a case mentioned by Elschnig[62]. The patient was a woman, 51 years of age, affected with icterus with enlarged liver and gallbladder, and she was also seized with keratomalacia shortly before she died. The autopsy showed a papilloma with a carcinomatously infiltrated base at the mouth of the bile duct with obstruction of the same, dilatation of the bile ducts and ascites. The patient died from pneumonia. In this case, where the obstruction of the common bile duct has completely stopped the flow of the bile to the intestine, the fats of the food and the fat-soluble A connected with them have scarcely been resorbed from the intestine, and here we obviously find the cause of the outbreak of the keratomalacia in this patient.

2. A. One of the very first experiences during the experiments upon animals and food-stuffs free from fat-soluble A, was that the animals did not grow upon this food, while on the contrary they immediately began to grow fast, as soon as the food was mixed abundantly with fat-soluble A. This may hardly be explained otherwise than that the growth itself consumes fat-soluble A; if this is abundantly added, the growth takes place—otherwise not. To the same effects the facts point that the xerophthalmia is much easier to develop in young rats than in adults, and that the keratomalacia is found in such an overwhelming degree in children just in the period when the quickest growth takes place. Consequently the need of fat-soluble A—in such a quantity as to prevent the xerophthalmia—must be greater during growth than in adults.

2. B. Likewise is the consumption of fat-soluble A surely increased during severe diseases—as was first supposed by Bloch and afterwards confirmed by Wright and Stolte. The fat-soluble A is most likely stored together with the spare fats of the body. During disease the spare fats, as is well known, are principally consumed, and it is an all but obvious conclusion, that the reserve supply of fat-soluble A is equally consumed to a great extent during severe diseases. To the same effects point the observation that xerosis conjunctivae connected with hemeralopia appeared principally with hard working persons, Kubli[31].

If these factors are taken into consideration it will be seen, that nearly all the cases of xerophthalmia mentioned in the literature may be explained as due to deficiency in fat-soluble A. While, as we have seen, some of these factors individually are able to develop the xerophthalmia, it is when two or more are combined, that we meet with the severer forms of disease. Here in Denmark—as will be seen later—it is principally the combination of 1. A. and 2. A. that has given occasion to the majority of the numerous cases of keratomalacia which appeared here during the last 12 years. There may possibly be some other—still unknown—circumstances that take

a part in the genesis of xerophthalmia, but those mentioned here are certainly the most conspicuous, as far as the Danish xerophthalmia is concerned.

OWN MATERIAL.

The material consists, as already said, in 434 cases of keratomalacia in children, 19 cases of keratomalacia in adults and 148 cases of xerosis conjunctivae without keratomalacia, all of them observed in Denmark during the years 1909 to 1920.

KERATOMALACIA IN CHILDREN.

The food. Concerning 295 of the 434 children affected with keratomalacia, information was given about the food given to the children before the disease broke out. The food was as shown by the following tabulation:

	Cases
Group I. Separator skim-milk or skim-milk + water	10
Elder children's food based on margarine (bread, margarine, potatoes, pork)	24
Barley, gruel, beer-caudle, oatmeal-gruel	64
Buttermilk-gruel	69
Skim-milk-gruel, separator-skim-milk-gruel	37
Oatmeal-gruel, barley-gruel + small quantities of milk, in the great majority of cases poor in fat	16
	220
Group II: Milk + water	12
Milk-gruel, oatmeal-gruel or barley-gruel + milk	61
Mother's milk	2
	75

As it will be seen, the 220 cases of group I had lived upon a food that was either completely deficient in fat-soluble A (barley-gruel, beer-caudle, oatmeal-gruel, etc.) or contained a tiny percentage of milk-fat and consequently only very little fat-soluble A (separator-skim-milk, skim-milk, buttermilk, etc.). In all these 220 cases of group I the food was therefore poor in fat-soluble A.

In group II were two children that had mother's milk, but as a matter of fact probably had scarcely any of it. One of them, a boy of 1½ months, had ever since his birth vomited his food. Besides, the mother was ill and had very little milk. Also, during the treatment the child went on vomiting; and in spite of a treatment with raw whole milk he would not thrive but died soon after. The other child was prematurely born and would not thrive either, so it probably did not get very much milk. Only in 22 of the other 73 cases of group II, we know with certainty that they had got whole milk. In the other cases nothing is remarked about the milk, so it may have been separator-skim-milk or skim-milk. Moreover the milk in most of the cases was given with abundant carbohydrates and had in many cases been boiled for some time, but nevertheless we may presume, that the food in the 22 cases had been relatively rich in fat-soluble A.

The food alone is not in all cases able to explain why the children got keratomalacia. But, as we have already said, other circumstances than the quantity of fat-soluble A of the food, take a part in the genesis of the xerophthalmia. The supply of fat-soluble A to the organism does not only depend on the food's content of fat-soluble A, but also on the manner in which the food is resorbed in the intestine, as digestive diseases may injure the resorption of the fat-soluble A in the intestine. Moreover, during a rapid growth and a severe illness the consumption of fat-soluble A is particularly great, and therefore these two circumstances will always predispose to xerophthalmia. All these facts considered, it will now be seen, that the keratomalacia in nearly all the 295 cases may be explained as due to the lack of fat-soluble A. In the great majority of these 295 cases the children were less than one year old, an age where the growth is extremely rapid. The food of these children was in 220 cases absolutely poor in fat-soluble A, and probably so in the great majority of the remaining 75 cases, but it is not out of the question that the food in some of the latter cases may have had a relative high content of fat-soluble A. If we consider these cases more closely it will be seen, that all these children in the time before the keratomalacia broke out had suffered from severe diseases—in most cases diges-

tive diseases; and this fact explains how these children, in spite of receiving fat-soluble A with the food, still had to suffer from deficiency of fat-soluble A.

All in all, these researches into the food given to the children before the keratomalacia broke out, have shown that it had, in the great majority of cases, been poor in fat-soluble A, and in many cases also in other respects poor in substances that are important for the child's nutrition, lack of which has proved active for the genesis of the xerophthalmia. In very few cases where it could not be proved that the food was poor in fat-soluble A, the child had been seized with diseases; which—according to what we know about the qualities of the fat-soluble A—perfectly explain why the child, notwithstanding the fat-soluble A found in the food, had to suffer from deficiency of fat-soluble A.

Duration of the food. If the keratomalacia is due to the child's having lived upon food poor in fat-soluble A, we might expect, that the less poor in fat-soluble A the food had been, the longer time might the child have been living upon the food before the keratomalacia broke out. But here evidently the age of the child enters into the question. In 123 cases where the food had been poor in fat-soluble A, we had information about the time in which the food had been given before the keratomalacia was established:

	Months	Months	Months	Months	Months
Age of the child.....	1-3	4-6	7-9	10-12	over 12
Average duration of the food	1,85	2,83	4,60	6,55	8,50

It will thus be seen that the food almost free from fat-soluble A of Group 1 developed the keratomalacia in shorter time than was the case with group 2 with a larger content of fat-soluble A in the food, and this group again represents a shorter time than group 3 with the food relatively well-provided with fat-soluble A. With the elder children the difference is not so obvious as with the youngest, but the elder children do certainly not live exclusively upon the food here mentioned, as the youngest do, but take now and then a cake, a bun, some milk or the like, so that the difference will be nearly effaced. *It is thus seen, that the food most poor in fat-soluble A in the shortest time provokes the keratomalacia, and that the youngest children live the shortest upon such a food, before the keratomalacia breaks out.*

Date of the appearance of the cases. For each case the date has been noted on which the doctor could diagnose the keratomalacia. This, of course, does not signify, in all the cases, the date on which the disease broke out, but it is not possible to indicate that moment quite exactly, and the difference will not be great in most of the cases.

This tabulation shows that the youngest children lived the shortest time upon the food poor in fat-soluble A, before the keratomalacia broke out.

TABLE I.

	Months	Months	Months	Months	Months
Age of the child.....	1-3	4-6	7-9	10-12	over 12
	Average duration of the food				
Group 1: Barley-gruel, beer-caudle, oatmeal-gruel	1,57	2,42	3,43	7,50	8,00
Group 2: Butter-milk, skim-milk, separator-skim-milk, milk-soup, etc.		3,12	4,70	6,20	9,00
Group 3: Gruel, sometimes with whole milk.	2,11	3,25	5,00	8,25	

TABLE II.

	1909	1910	1911	1912	1913	1914	1915	1916	1917	1918	1919	1920	Total
January...	1	3	1	5	3	4	2	7	14	4	0	3	47
February .	4	2	3	9	8	3	2	13	7	0	0	0	51
March	1	4	10	7	6	11	19	11	12	2	1	4	88
April	4	4	1	8	7	8	12	21	13	1	1	4	84
May	3	9	2	7	6	4	8	10	15	2	0	9	75
June	3	1	1	1	2	2	2	1	5	0	0	1	19
July	1	1	0	0	3	2	0	0	2	0	0	2	11
August ...	1	0	0	0	3	1	1	3	2	0	0	0	11
September.	1	0	0	1	0	3	2	1	0	0	0	0	8
October...	0	0	0	1	1	1	0	1	0	0	1	1	6
November.	0	1	1	0	1	3	3	3	1	0	0	0	13
December.	1	1	1	1	0	6	2	7	0	0	1	1	21
Total	20	26	20	40	40	48	53	78	71	9	4	25	434

The above tabulation shows how the cases are distributed in months and years thruout the twelve years. It will equally be seen from the last horizontal line in Table II how the cases increased during the years 1909 to 1917, how in 1918 and 1919 a sudden falling-off took place, and then again a rise in 1920.

The last vertical column of the table II and Fig. 2 shows that the cases are not equally spread over all the months of the year, but that the number increase during the winter months and reached the maximum in March. In April and May appear many cases, but then there is a sudden downfall in June and July, and August, September and October only show a few cases.

This distribution of the cases over the winter and spring months, which appears very markedly when all the cases are seen together, is also to be found if each year is considered by itself. There are, however, small differences in the distribution in every single year, the maximum arriving some years earlier than in others. In order to show in relief these differences and obtain an exact curve representing the distribution of the cases all the year round thruout the 12 years, the curve 3 has been drawn.

This curve illustrates the frequency of

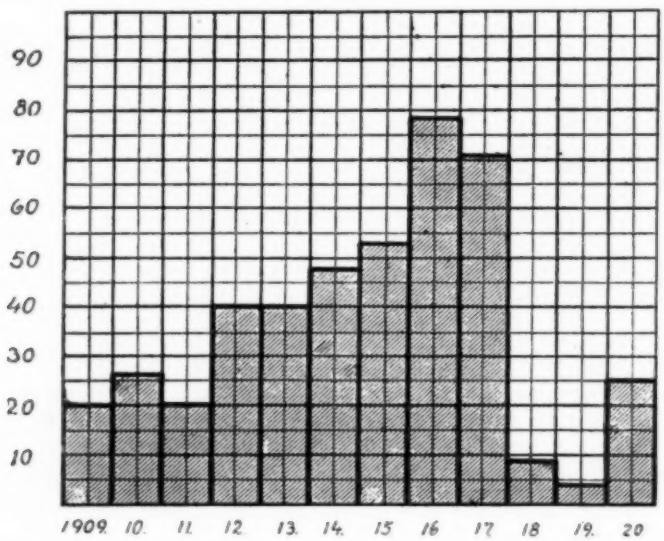

Fig. 1. Number of cases of keratomalacia occurring each year from 1909 to 1920, numbers at left, years at bottom.

the cases thruout the period, calculated as explained in my monograph,[200] on pp. 81 to 84. As unit for the time, indicated on the figure as abscissa, is used 1/360 of a year or practically one day.

Thus the curve illustrates the frequency of the cases of each single date thruout the 12 years. We here observe

How can we explain these variations in the frequency of the cases of keratomalacia in Denmark during the years 1909 *to* 1920? These variations are so important, that we must absolutely suppose a corresponding variation in the children's supply of fat-soluble A during the 12 years, if our theory shall fit.

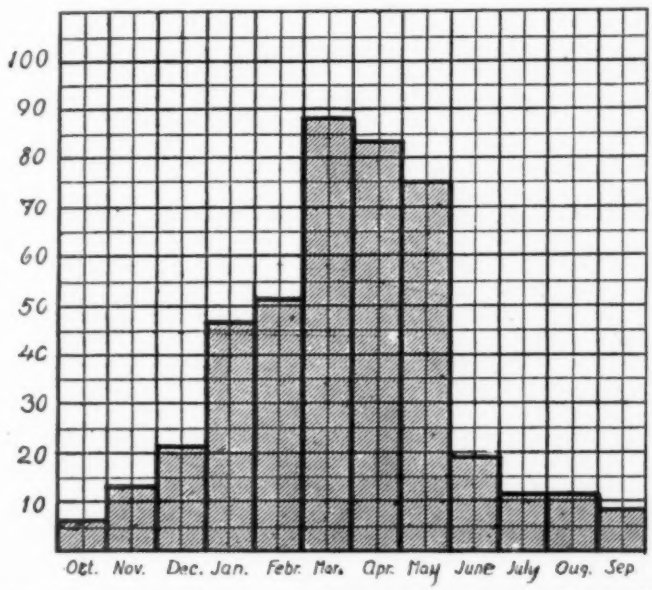

Fig. 2. Distribution of cases by months, fewest in October rising to most in March with a sudden drop in June.

both the increase of the frequency during the years 1911 and 1917, and more especially the increasing frequency during the winter and spring months and the sudden decrease towards summer. It is equally seen how this decrease in some years occurs early in the year, in others later.

The result of the preceding observations are as follows:

1. There was a strong gradual increase in the number of cases of keratomalacia in Denmark during the years 1911 to 1917.
2. Then came a sudden fall during the years 1918 and 1919.
3. Then another increase in 1920.
4. The cases of keratomalacia appeared principally during the winter and spring months (December to May).
5. The majority of cases occurred in March and April, but the maximum was not attained at just the same date every year.

How can children's supply of fat-soluble A have varied during these years? The fat-soluble A is supplied exclusively by the food and cannot be formed by the animal organism. If young rats do not get fat-soluble A with their food, they will die. But not immediately; they are even able to grow a short time, before they fall off in weight. The reason for this is that the animal organism contains normally a reserve of fat-soluble A. If the food does not contain any fat-soluble A, the reserve fat-soluble A is consumed, and the animal will only grow until this reserve has been exhausted. Even newborn rats possess normally a reserve fund of fat-soluble A, if the mother's food has contained a quantity of this substance large enough to supply the fetus with it during gestation. If this was not the case, the young rats died shortly after the birth, their reserve of fat-soluble A being too small.

Later on the young ones store up fat-soluble A as reserve, if their food contains more than they consume for the sustenance of life and growth.

Just the same happens with the child. It is born with a reserve fund of fat-soluble A—the quantity of which varies according to the mother's food during the gestation. Later on this reserve is either consumed, balanced or increased, according to the content of fat-soluble A of the child's food. The children generally get their mother's milk at first (in nearly all the cases where the food is mentioned, the children have nursed for a shorter or longer time). The content of fat-soluble A of the mother's milk may also vary—namely according to the content of fat-soluble A of the mother's food. If the mother's food is quite insufficient in this respect, even sucklings are exposed to keratomalacia, as demonstrated by Thalberg, Mori and Schiele. Consequently the mother's food takes a part in the child's supply with fat-soluble A.

The artificial nourishment which the children seized with keratomalacia had after having ceased to nurse, consisted either in food completely destitute of fat-soluble A (oatmeal-gruel, barley-gruel, beer-caudle, etc.) or it derived its content of fat-soluble A almost exclusively from cow's milk. As we already mentioned, the content of fat-soluble A in the cow's milk varies with that of the food. If the cows are "stall fed," the content of fat-soluble A is less, than if the cows were on pasture. This depends undoubtedly on the fact that the stall food is poorer in fat-soluble A than the grass food. Grass contains, even in its dried condition, as hay, a good deal of fat-soluble A; while on the contrary cattle-beets and forage cakes—especially forage cakes prepared from vegetable seeds such as linseed, sunflower seed, cotton seed, soya beans and the like, do not contain much fat-soluble A.

The child's supply with fat-soluble A depends in these cases on:

I. The content of fat-soluble A in mother's food.

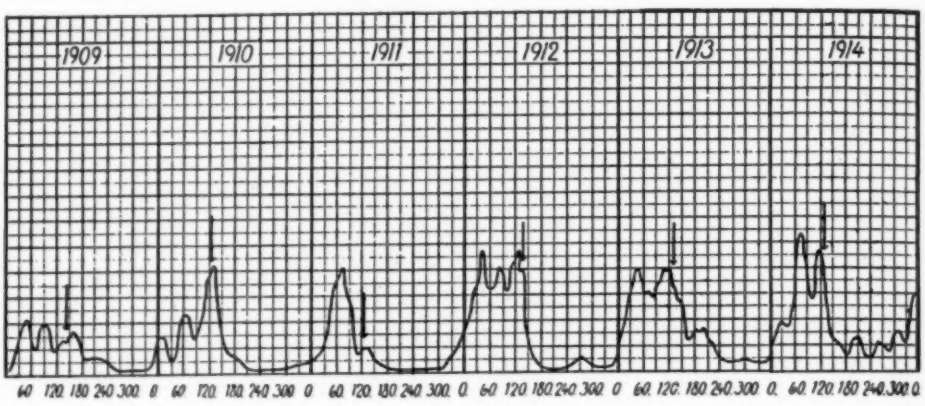

Fig. 3. Distribution of cases in each year

II. The content of fat-soluble A in the cow's food.

Have there now been changes in I and II, which may explain the above mentioned fluctuations in the frequency of the cases of keratomalacia?

I. The adult persons receive principally their fat-soluble A from the fat of the milk and especially from the butter. In former times all adult persons in this country ate rather big quantities of butter; recently, however, the butter has been supplanted by the cheaper artificial butter, the margarine. This stuff is particularly employed by the economically less well circumstanced people, to which belong, as will be seen afterwards (See: *The situation of the parents*), nearly all the children of this study. In the beginning the margarine was prepared from animal fats and consequently contained certain quantities of fat-soluble A, according to the content of fat-soluble A of the fats employed. The content of fat-soluble A in the animal

margarine, however, is not so great as that of butter (shown by experiments on animals carried out by Steenbock, Boutwell and Kent[160]. But during later years, the vegetable margarine has more and more supplanted the animal one; because it can be produced cheaper and has a better taste than the animal margarine. The vegetable margarine contains but very small quantities of fat-soluble A, as it is prepared from fats destitute of fat-soluble A. Even when the margarine is churned with some milk, its content of fat-soluble A, as shown by insignificant quantities of animal raw materials. The increase of the consumption of the margarine is due to its more moderate price and steadily improved quality. The decrease, on the contrary, is owing to the blockade during the war, which rendered the importation of raw materials for margarine nearly impossible. These raw materials must now first be prepared, the oils must be pressed out, purified, etc. and finally prepared as margarine. All these operations take a certain time, so that a curve illustrating the margarine production (unfortunately

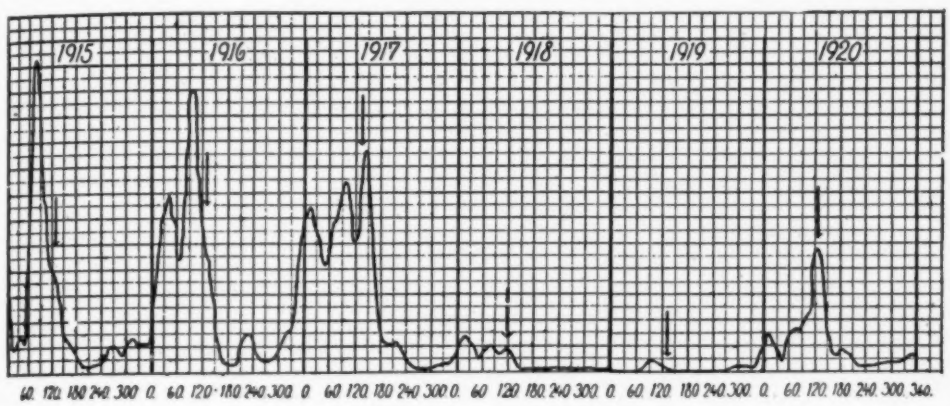

Fig. 3, 2nd part. Number of years at top. Days at bottom.

Halliburton and Drummond[189] and later by Drummond[151], is quite insufficient.

The curves indicate for each year the total quantity (in millions of kg.) of margarine (vegetable and animal) and butter consumed here in Denmark.

The black columns indicate the importation of raw materials for the manufacture of vegetable margarine for national consumption. The white columns indicate the importation of raw materials for the manufacture of animal margarine and the shaded columns indicate the consumption of butter.

As will be seen from figure 4, the importation of raw materials for the manufacture of vegetable margarine for national consumption increased very considerably during the years 1909 to 1916, simultaneously with the decrease of the importation of animal raw materials. In 1917, and especially in 1918, very small quantities of vegetable raw materials were imported, but in 1919 and 1920 again considerable quantities and only it was not possible to obtain one) should be supposed to be placed rather farther into futurity.

The consumption of butter stands, as seen from fig. 4, in inverse proportion to that of margarine. These two foodstuffs replace each other in the household. If margarine is used on the bread, for cooking, baking and the like, we will do without butter. As the manufacture of margarine at the end of 1917 ceased, on account of the import of the raw materials being rendered impossible by the submarine blockade, the butter in Denmark became so expensive that a good many of the poor people could not afford to use it. But after the 21st of December, 1917 the butter was rationed, so that everybody could buy 250 gr. a week at a price so moderate, that even the less well-to-do people could afford it. In the period from the 21st December 1917 to the 1st May 1919 (when the rations ceased), everybody in Denmark ate butter, and after this date the manufacture of mar-

garine began again, and this food-stuff began again to supplant the butter on the table of the less well-to-do people.

It will thus be seen that the increase and the falling off in the consumption of margarine and the corresponding falling off and increase in the consumption of butter in Denmark are in direct correspondence with the increase and the falling off of the cases of keratomalacia. There can be no doubt that this change in the food, from margarine to butter and vice versa, has taken a very great part in the daily supply of fat-soluble A in those not well off. There can therefore be no doubt that the food of the adults during the years when the cases of keratomalacia were rising contained less fat-soluble, A, and that it contained more fat-soluble A during the years when the cases of keratomalacia were decreasing.

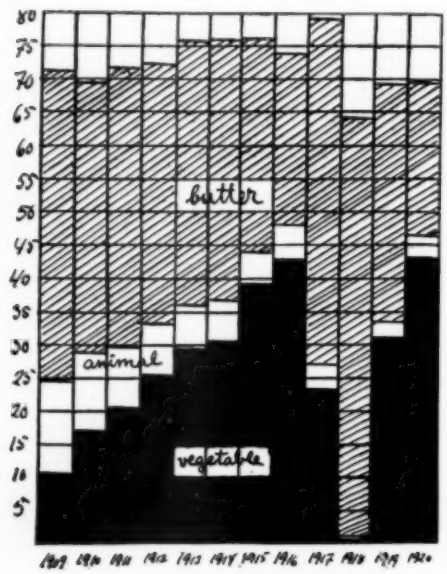

Fig. 4. Relative amount of butter used each year (oblique lines); substitutes made from animal fats (white spaces); and vegetable fat substitutes (black spaces).

The quantity of fat-soluble A in the mother's food has thus varied in the said period, and this fact has been important for the children's supply of fat-soluble A during the period. For when the content of fat-soluble A in the mother's food was relatively small, as for instance in 1916, the reserve fat-soluble A born with the children in that year was also relatively small. Also the mother's milk was in that year relatively poor in fat-soluble A, and the child's reserve fat-soluble A kept on relatively small. If the child now gets food poor in fat-soluble A, the reserve will rapidly be exhausted, and the keratomalacia will appear. If the reserve had been larger, the child might have endured a short "wrong" nourishment without being seized with keratomalacia. We may thus explain the great number of cases in 1916.

Other circumstances, however, have certainly taken part in the varying distribution of the cases of keratomalacia during the years 1909 to 1920. Far the greatest number of cases appeared among the people less well off. These people had their income considerably increased during the years 1918, 1919 and 1920, as the wages increased rather much in Denmark during these years. The increase exceeded considerably the price index in this country. These people were thus much better off during the years 1918 and 1919 than during the years before and after, and were able to afford the more expensive food-stuffs that were richer in fat-soluble A. This has most likely been of importance for the decrease of the xerophthalmia during these years. Finally the high tax on spirits introduced just in these years in Denmark has diminished the abuse of alcohol in a considerable way, and thus bettered the fortunes in many poor homes. There is also a reason to believe that the work of Prof. Bloch about xerophthalmia published in 1917, a work that called the attention of the Danish physicians to that disease, deserves a great part of the credit of having contributed to the rapid decrease of the cases of xerophthalmia during the years 1918 and 1919. It is very likely that a number of the children who showed incipient symptoms of xerophthalmia were saved from the keratomalacia by the change of diet ordered in time by the doctor.

That xerophthalmia appears preferentially during the spring months had been known previously. It had especially been observed that xerosis conjunctivae with hemeralopia appeared most frequently in the spring (Schtschepaatjew[58], Trantas[72], Generopitomzew[80], Kreindel[89], Wietfeldt[131], Zak[147], especially during the Russian Lent, Blessig[6], Thalberg[25] and Kubli[81]). But in England Stephenson[61]

observed the disease most frequently during the summer and autumn months, and he also found, that keratomalacia appeared most frequently in the summer and in the months of enteritis[101]. The same experience is mentioned by Hirschberg[11], Leber[24], Herbert[58] and Mori[82], while Schiele[92] and Gille[113] observed far the most cases of keratomalacia in the spring months. Finally the cases of keratomalacia observed by Kapuscinski[108] are equally distributed over all the 12 months of the year. The cases appearing during the summer months, as already mentioned and pointed out by several authors, relate to the children's enteritis, very frequent in these months.

Here in Denmark, as shown first by Rönne[127], later by Bloch[149], keratomalacia appears by far most frequently during the winter and the spring months. Bloch has pointed out how this may be explained by the fact that the growth of the children is most rapid in the spring months, and as rats do not grow if their food is deficient in fat-soluble A, we must suppose that fat-soluble A is consumed during the growth. If, therefore, especially in the spring months a great quantity of fat-soluble A is consumed, we understand that children getting food poor in fat-soluble A are most frequently seized with xerophthalmia just in that season. Many reasons seem to favor this supposition, even if the curves of growth and xerophthalmia do not perfectly run together, as the maximal period of the growth of height does not stop until the middle of August—2½ months after the great falling off of the xerophthalmia curve (this fact will be explained later on). The theory, however, that fat-soluble A is consumed during the growth, is so well founded that the coincidence between the period of the most rapid growth and the maximum of the cases of xerophthalmia speaks greatly in favor of the correctness of the explanation.

Saunte[159] observes that the disappearance of xerophthalmia in the months of May and June might be due to the fact that the cows are on grass in that period. During the winter the cows' milk was getting more and more poor in fat-soluble A, on account of the stall feeding poor in this material, and consequently more cases of xerophthalmia appeared; but about the middle of May the cows began to eat grass, rich in fat-soluble A, the milk became again rich in fat-soluble A, and no more cases of xerophthalmia appeared. The experiments on animals are indicative that this is the right explanation. Maybe the fresh green grass in the spring is especially rich in fat-soluble A. The cases of xeropthalmia cease to appear just in the period when the cows in Denmark are driven on grass.

On the curve of frequency (fig. 3) we find marks that indicate for each year the date when all the cows are generally driven on grass. As it will be seen, the date when all the cows were driven on grass, for the 12 years coincides nearly exactly with the date when the curve of frequency shows its great falling off in the corresponding year, notwithstanding that this date is not the same every single year. There is, however, some difference in the interval between the last top of the curve of frequency and the date of pasture, but this date, of course, cannot be quite exact as in some parts of the country the cows are driven on grass several weeks before they are driven on grass in other parts of the country, and when we here as the date of pasture indicate the date when the cows all over have generally been driven on grass, it may be supposed that in several parts of the country the cows have already been grazing for some time. The milk which is gathered from many different farms will therefore at an early date be added with some milk containing fat-soluble A, and it may thus be supposed that the curve of keratomalacia will begin to decline some time before the date of pasture, as it really does. The different dates for the ripeness of the grass in the different parts of the country may explain, that the date of pasture does not every year lie at the same distance from the top of the curve. But in spite of these differences the date of pasture and falling off of the curve coincide remarkably, even when both vary from one year to another. In order to illustrate the conformity between the date of pasture and the drop of the curve of frequency of each year in a clearer way, than it may be seen on fig. 3, we have drawn fig. 5, on which for every year the date (expressed by its

number in the year calculated at 360 days) when the pasture began is marked with a cross, and the fall of the curve of frequency is illustrated by means of a piece of line from the date of the beginning of the fall to that of the end. The ends of these pieces of lines are connected with lines, and the crosses are again connected with a stippled line, and

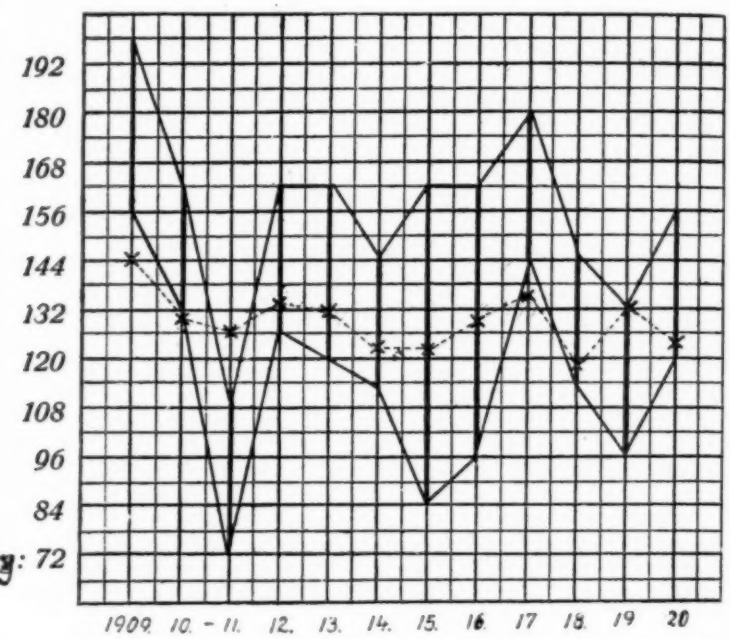

Fig. 5. Relation of turning cows out to pasture and drop in keratomalacia. Cross marks date of pasture, heavy vertical line drop in cases. Left figures day of year, bottom number of year.

it will now be seen how the movements of this line thruout the years follow those which mark the fall-off of the curve of keratomalacia. The direction of the movements are during all the years the same for the stippled line and the figures. Only in 1919 it is different; but in that year only 4 cases of keratomalacia occurred. Here accidental circumstances take a great part, so that a divergence in that year has no importance at all.

The figure illustrates thus, that in the years where the date of pasture occured early compared with other years, in the same years the cases of keratomalacia ceased earlier than ordinarily—and vice versa.

It must be supposed, it is true, that these variations in the content of fat-soluble A in the cow's milk are only very small, and under ordinary circumstances they surely are of no importance. But upon small children, whose food has been poor in fat-soluble A, and who therefore are in the "twilight zone" as to fat-soluble A, they certainly may have some influence for the eventuality whether fat-poor milk contains just enough fat-soluble A, or not, to prevent children, after having been nourished a long time with such food, from being seized with xerophthalmia.

It will thus be seen, that we are able to explain in this way, why keratomalacia in Denmark appears particularly during the winter and spring months; but it is highly probable that also other circumstances take a part in this fact. Not only xerophthalmia but also other diseases appear with special frequency during the winter and the spring, for instance rheumatic and catarrhal diseases, which are supposed to depend, somehow or other, on the meteorologic circumstances, and the same reasons might be extended to xerophthalmia. And finally so many reasons favor the theory of Bloch about the importance of growth, that we must presume, that the faster growth during the spring months takes part in the accumulation of the cases of

keratomalacia in these months. As we have already seen, the child is growing (according to the investigations of Malling-Hansen) most rapidly from April until quite a part of August; and, consequently, several months after the culmination of the curve of keratomalacia. It is reasonable to suppose, that it is the increasing content of fat-soluble A in the milk, after the cows have been driven on grass, which prevents new cases of keratomalacia from breaking out, in spite of the fact that the maximal growth is still going on.

Thru the investigations of the variations in (I) the content of fat-soluble A in the mother's food, and (II), the content of fat-soluble A in the cow's food, we have thus succeeded in explaining the variations in the curve of frequency of the cases of keratomalacia.

It will thus be seen, that all the variations in the frequency of the cases of keratomalacia thruout the 12 years, which this study includes, may be explained; not only by the theory that the xerophthalmia is due to the lack of fat-soluble A, but that they directly speak in favor of the accuracy of the theory.

THE AGE OF THE CHILDREN.

While the xerosis of the conjunctiva with hemeralopia most frequently is observed in elder children, keratomalacia is distinctly a child's disease. The affection of the cornea with the xerophthalmia is very seldom seen in adults, and the typical sudden necrosis of the whole cornea is only observed in children. Keratomalacia attacks most often children younger than one year. The babies (of about 1 month) are however seldom attacked with this disease, as already observed by v. Graefe. The disease is here in Europe also infrequent with elder children. Saemisch[83] indicates the first year of life as the most exposed, Flemming[95] the first 9 months of life. Among the 92 cases observed by Gille[113], 65 were from 1 to 6 months, 14 from 6 to 12 months, and only 7 more than a year. In 6 cases no age was indicated. Among the cases observed by Kapuscinski[108] the figures were 17, 5 and 9 for the corresponding groups of age. Stephenson indicates that the maximum is at the age of 8 months and Gille at 3 months.

While the keratomalacia in Europe seems to attack principally small children, the communications of Gama Lobo[7], Teuscher[9], de Gouvea[ ]nd Mori[82] show that keratomalacia in A erica and Japan attacks also frequently 'er children.

The following tabulation illustrates the distribution of age among the 430 cases of keratomalacia (in 4 cases no age was indicated).

TABLE III.

	Cases
1 month	3
2 months	19
3 months	29
4 months	33
5 months	41
6 months	52
7 months	36
8 months	27
9 months	38
10 months	23
11 months	23
12 months	44
13 months	8
14 months	5
15 months	7
16 months	3
17 months	0
1½ years	11
2 years	12
3 years	5
4 years	4
5 years	1
6 years	2
7 years	2
8 years	1
9 years	0
10 years	1

This tabulation however, does not give the exact idea of the real distribution of age. A child of 8 or 10 months is often indicated by the mother as being 3/4 year of age and is accordingly registered in the 9 months' group, which, therefore, becomes too numerous. This may be said in a still higher degree about the groups including children of 1 to 1 1/2 years. On the following fig. 6 the curve has been corrected for these errors by merely graphic interpolation on the curve of the summing-up-function, which afterwards has been recalculated to an ordinary curve of frequency.

As illustrated by this curve, the top point lies in the age of 6 months, con-

sequently the age when children here in Denmark are most often seized with keratomalacia. Still the whole first year of life, except only the first month of life, is particularly exposed, tho the disease quite frequently may be observed in children up to 2 and 3 years, and on rare occasions even in adults (see later).

As far as we can judge from the experiments on animals, the tendency to get xerophthalmia on account of food poor in fat-soluble A is greatest with the youngest children and declines with increasing age. After the analogy with this fact, infants are especially exposed to the keratomalacia, and when the very youngest groups do not count as many cases as the group of about 6 months the reason is certainly, that the disease does not break out until the child for a certain time (2-3 months for the youngest, Table I. page 95) has been living upon the food poor in fat-soluble A. For the decline of the keratomalacia with increasing age it is also certainly of importance, that the food of the elder child is more varied and consequently less frequently thoroly poor in fat-soluble A for a long time.

The situation of the parents. The keratomalacia is most frequently observed in poor homes. Among the cases of the material in question the parents' situation was: in 137 cases workmen, in 87 cases unmarried mothers, in 68 cases artisans and in 21 cases cottars.

THE TWO GROUPS OF THE MATERIAL.

The numerous particulars about each single case (date and year for the outbreak of the disease, age, domicile, situation, food, etc.) has rendered it possible to group the material in many different ways. A series of groupings, which on account of lack of space must be omitted here, have resulted in establishing[200] two groups of cases in the material: 1. Small children (1 to 4 months) who had got food rich in carbohydrates. These cases did not appear so particularly during the winter and spring months as the rest, and occurred especially in the towns. 2. The others whose food had been in a great measure poor in milk-fat, who lived chiefly in the country and who showed the distribution typical for the Danish cases: their appearance especially during the winter and spring months. The first group, that probably corresponds with the atrophia and dystrophia amylogenetica pointed out by Bloch, and with the cases of xerophthalmia observed also in other countries together with "Mehlnährschaden," includes small children living upon food very rich in carbohydrates and where diseases, especially digestive diseases due most probably to the food rather inadequate for children of that age, take a great part as predisposing causes for keratomalacia —partly thru the increased consumption of fat-soluble A, and partly thru the deficient absorption of fat-soluble A from the intestine. In these cases the content of fat-soluble A consequently does not take so great a part, for which reason the grouping of the cases is less influenced by the variations in the content of fat-soluble A of the food-stuffs.

The other group, which corresponds probably to the dystrophia alipogenetica mentioned by Bloch, includes older children who had lived upon milk-food

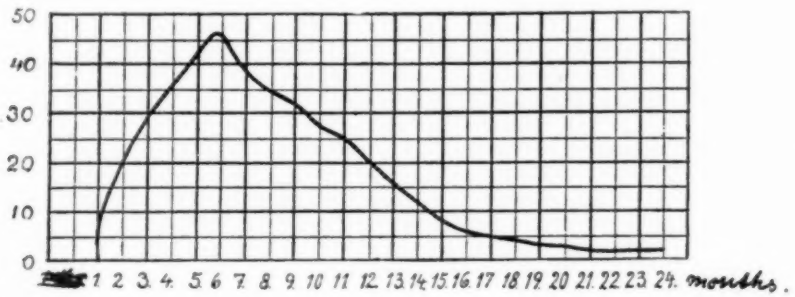

Fig. 6.—Age at which the disease occurs. To the left number of cases, below age in months.

poor in fats, where the variations of fat-soluble A of the food take a great part in the genesis of the keratomalacia; because it is first and primarily the lack of this substance that renders the food inadequate to the child, and because the supply of fat-soluble A thru this food most probably is on the borderline of the minimum of

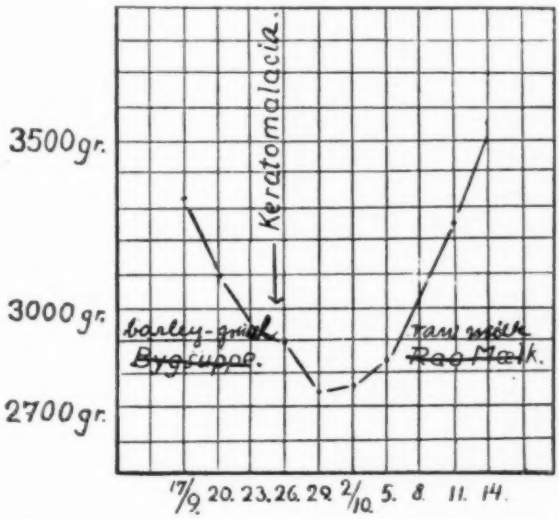

Fig. 7. Loss of weight preceding keratomalacia. Weight on left, day of month below.

fat-soluble A, indispensable for preventing keratomalacia. This is the reason why the variations in the content of fat-soluble A of the food play such a great part in the disposition of the children to keratomalacia. The latter group of cases is far the greatest as far as Denmark is concerned and is the prominent feature of the whole material. In other countries where the dairying and farming are less developed than here, this group is surely wanting and the disease will there be far more infrequent than here.

Loss of weight before the outbreak of the keratomalacia. In conformity with the experiments on animals, there will surely be observed a loss of weight in the children in the time before the keratomalacia breaks out. In 6 cases, where the keratomalacia appeared during a stay in the hospital, a loss of weight, or a standstill in the time before the disease broke out was observed, as will be seen from the weight-curve on fig. 7-12. It will be observed, that the curve as a rule declines a little at first after the beginning of the milk-diet. As a rule, a short time will pass before the milk-diet begins to act. But in some cases—where the food was rich in carbohydrates—the loss may be rather considerable, and is here due to increased secretion of water. In these cases the diuresis will increase during the first days after the beginning of the milk-diet, as the organism rids itself of the unnecessary liquid which has been bound in the pasty, inflated subcutaneous tissue. The curves illustrate in all the cases the fair increase of weight after the treatment with milk.

In 50 cases the weight of the child when it was removed to the hospital was indicated. These were all children of from 2 to 15 months of age. In nearly all these cases the weight was about 200 grams under the normal weight for children of the same age. The children were deficient in weight at the outbreak of the disease, consequently they must have suffered a loss of weight, or they had grown too slowly during the time before.

PROGNOSIS AND MORTALITY.

While the xerophthalmia in adults is cured spontaneously without prejudice to the future, if the cornea, on very rare occasions, has not been attacked, the conditions are quite different with children.

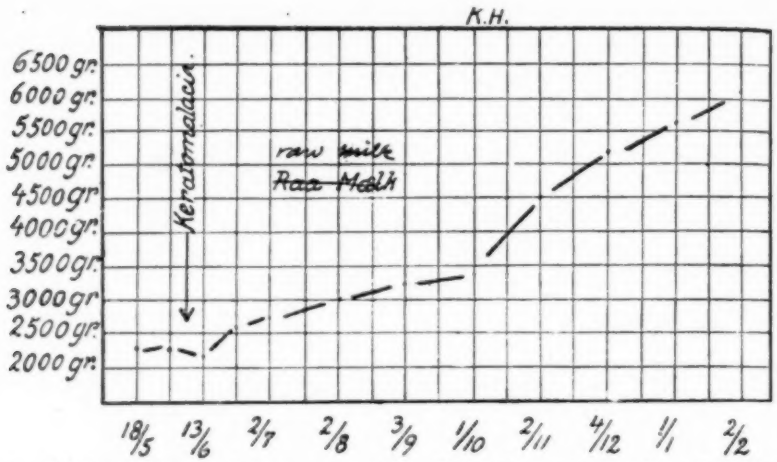

Fig. 8. Gain in weight after feeding on raw milk. Weight in grammes to left. Dates below.

In older times keratomalacia in children was regarded as a most severe disease and even as a symptom of an imminent death. We understand that, when we know that mostly the cases observed in those days proved fatal, for instance, all the cases observed by v. Graefe[8] and likewise all the 32 cases registered by Hirschberg[11]. Leber[24] regarded it as a rare case if a baby survived the disease, and this is also advanced by Braunschweig[38] in 1890. As late as in 1903 Fehr[77] notes as a curiosity a case of keratomalacia in a baby, whom he succeeded in saving from death and conserving the faculty of vision. In the text-books and manuals (Fuchs, Roemer, Foerster, Groenouw, Goldzieher), this disease is therefore indicated as having a bad prognosis in babies. While thus the oldest statements show nearly a 100% of mortality, the prognosis of the later statements is somewhat more favorable. Of the 18 cases registered by Knaebel[70] only 9 died (mortality 50%); Stephenson[101] had likewise among 31 cases a mortality of about 50%. Of the 30 cases mentioned by Flemming[95] only 13 died (43%), Gille[113] mentions 92 cases with a mortality of 50%, and nearly the same mortality is found with the 31 cases of Kapuscinski[108] of which 14 died.

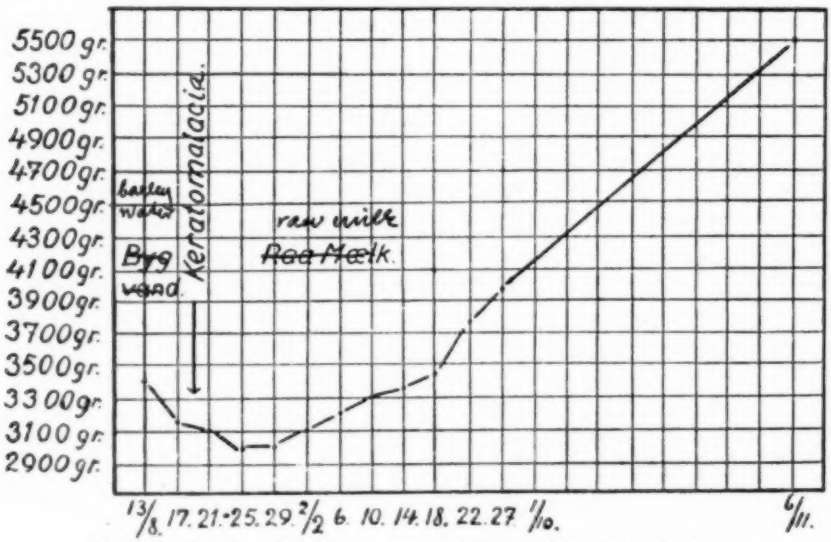

Fig. 9. Relation of weight changes to keratomalacia and to feeding.

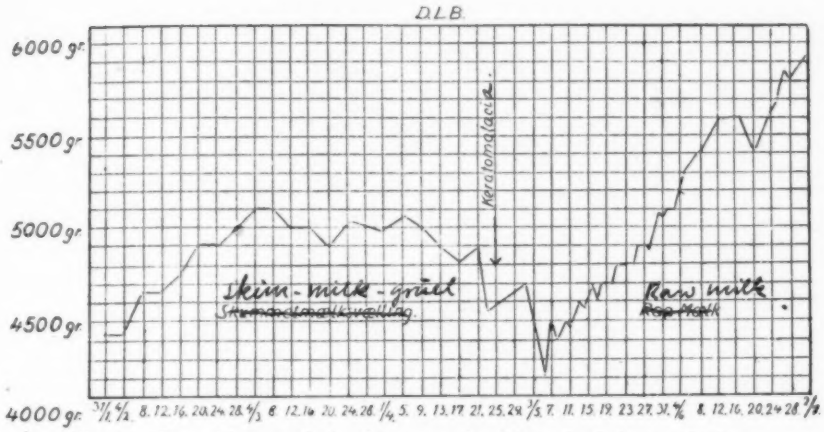

Fig. 10. Case of keratomalacia with curve of weight and relations to feeding.

The average mortality may therefore be regarded as 50%. While thus the prognosis for the babies was very bad, various authors (Fuchs, Roemer, Goldzieher) point out that it is considerably better with the somewhat older children.

Here in Denmark, where the milk treatment has been known and practised for a longer time, it might be expected that the mortality for this disease should be considerably less. Among the 434 cases of keratomalacia included in this material 87 died during the disease, while 6 were requested to be dismissed or evaded treatment in a dying state and must, therefore, be supposed to have died. If these 6 cases are counted, the percentage of mortality will be 21.5. Tho this figure is low compared with the former and therefore speaks highly in favor of the treatment with milk, the percentage of mortality of Bloch, only half the size of this, shows that the figure can be much lower with the treatment in a hospital (the great majority of the cases of this material have been treated out of bed) with milk-fat and cod liver oil.

As it will be seen from the above, the mortality is greatest among the youngest children and declines with the increasing age. No child more than 3 years of age died from the disease. This is in perfect conformity with the experiments on animals, that showed, that fat-soluble A (and also water-soluble B) is most indispensable to the youngest individuals,

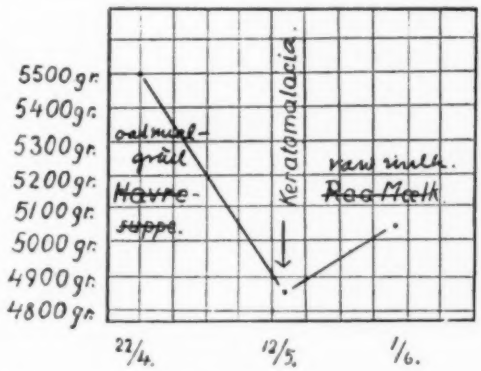

Fig. 11. Comparison of effects of oatmeal-gruel and raw milk.

who most quickly succumb to the disease (Drummond[152]).

Concerning 84 of the children who died from keratomalacia we know exactly the date of the decease. We have thus been able to calculate the time that passed from the date when the disease was established and the date of the decease.

TABLE IV.

	1-3 months	4-6 months	7-9 months	10-12 months	More than one year
Number of dead	20	28	25	12	7
Cases	51	126	101	90	62
Mortality	39%	22%	25%	13%	11%

TABLE V.

	1-3 months	4-6 months	7-9 months	10-12 months	More than one year
Total number of days.....	290	248	228	45	23
Number of cases.....	19	25	25	9	6
Average number of days.....	15	10	9	5	4

The above tabulation shows the calculation of the number of days that passed, for the 5 groups of different age between the date when the keratomalacia was established and the date of decease. We here observe the curious fact, that in the elder children death occurred a shorter time after establishing the discase than in the younger ones. The reason must certainly be, that the affection of the cornea (as a rule it is the serious symptoms of this affection that brings the child to the doctor), in the younger child appears earlier during the disease than in the older ones. The vulnerability of the cornea declines with the increasing age, so that it may easily be supposed, that the necrosis of the cornea with the somewhat older children does not appear until the last stage of the xerophthalmia—shortly before death. The bigger the child is, the less is the danger that the xerophthalmia will give lesion of the cornea; in adult persons the xerophthalmia is indeed extremely rare.

The cause of death is nearly always the bronchopneumonia. In 20 cases of this material section had been made, and in 16 of these cases bronchopneumonia was established. There was in 9 cases purulent bronchitis, in 6 cases enteritis, in 6 cases infantile atrophy, in 3 cases hepatic fatty degeneration, in 2 cases hepatic steatosis, in 2 cases pyelitis, in 2 cases cystitis and in 1 case nephrolithiasis. In 60 of the mortal cases we had information about complications. Among these bronchopneumonia was established in 46 cases.

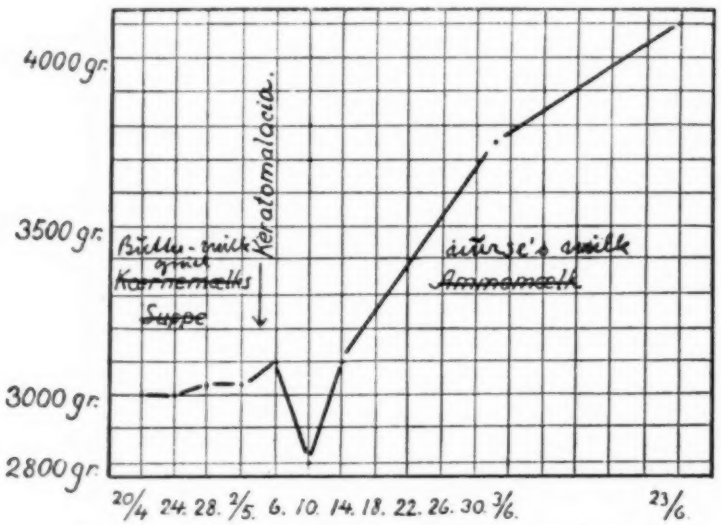

Fig. 12. Comparison of buttermilk and gruel, with nurse's milk.

THE PERCENTAGE OF BLINDNESS.

In 391 of the 434 cases we found information about the state of the eyes at the end of the treatment. As already said 93 succumbed to the disease. Of the remaining 298 cases, 79 (27%) were blind, and 71 (24%) had vision greatly reduced in both eyes. In 105 cases (35%) the result was a greatly reduced visual acuity in one eye, but good vision in the other; and, finally, in 43 cases (14%) recovery of both eyes without any reduction of the visual sense.

Altho the treatment with milk in the cases of this series, as already said, showed its efficacy thru a much lower

percentage of mortality than otherwise in the case with the infantile keratomalacia, the above remarks have shown how dangerous keratomalacia is for small children. Among the children who survived the disease, 27% were blind, 24% nearly blind and 35% lost the vision completely or almost completely of one eye. Only 14% recovered from the disease with fairly undamaged vision. This lamentable result is due principally to a treatment initiated too late; for what here has once been lost the treatment can never restore. The majority of the children were submitted too late to skillful medical treatment. The xerophthalmia shows in the beginning so few alarming symptoms to the nonprofessional, that the child is not brought to the doctor. It is not until the affection of the cornea appears and gives its symptoms that the doctor is called. And not unfrequently the affection of the cornea is not noticed immediately, because the child keeps the eyes shut and the reaction on the part of the eye very often is completely wanting. As moreover the affection of the cornea is proceeding with such a rapidity, that in the course of a few hours the cornea may necrose, it will be understood, that many of the children were not submitted to medical attendance until the corneæ were totally necrosed and the sight could no more be saved.

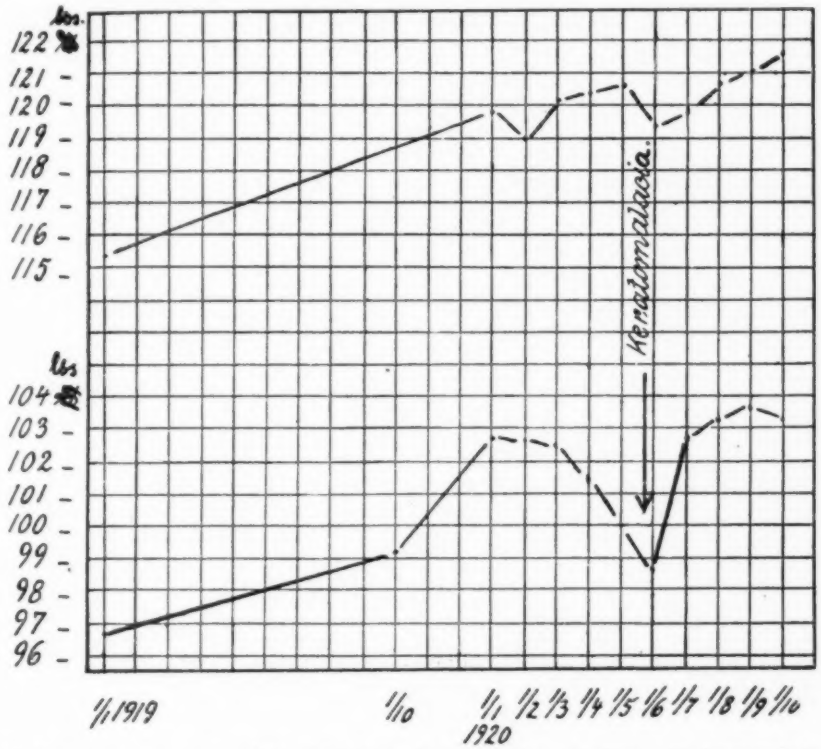

Fig. 13. Upper curve average weight of inmates. Lower weight of patients with keratomalacia. Left pounds, below dates.

Still the pecentage of blindness is less here than in statistics of previous dates. Among the 18 cases mentioned by Knaebel[70] only 9 survived the disease. 13 eyes were attacked of which 11 became completely blind (85%). Schiele[92] communicates 33 cases of keratomalacia; but only as far as 22 are concerned do we know the results. Of these 13 were blind (59%), 7 were blind in 1 eye and two had maculæ. Flemming[95] indicates, that in 4/5 of his cases there appeared large, frequently adherent leucomas, and that in nearly all the cases both eyes were attacked. Kapuscinski[108] observed 24

of his 31 cases after the disease had ceased. Of these 15 were blind (63%). In 9 of them the cornea was cured without interfering with the sense of sight. In 24 children the affection was bilateral. Stephenson[101] indicates, that half of the children who survived keratomalacia got blind, and Gille[113] says that in 42 children who were dismissed as recovered 46 eyes were blind (55%). In 74% of the cases both eyes were attacked. As it will be seen, the percentage of blindness in all the statistics here mentioned lies between 50 and 85. It is equally seen, that in about 3/4 of the cases the affection was bilateral. The percentage of blindness in our material was 27, so that even if 24% had the sense of sight greatly reduced in both eyes, the results still seem better than that of the former statistics.

As already mentioned, it is not always both eyes that are attacked with keratomalacia, and more especially they are seldom attacked at the same time. Generally a week passes between the appearance of the disease in the first and in the second eye. Among the cases of our material the right eye only was attacked in 43 cases (14%), the left eye only in 57 cases (19%), while both eyes were attacked in 198 cases (67%). The great number of bilateral keratomalacia seems to show how late the majority of the children have been submitted to medical treatment; for altho it may happen, that the other eye is attacked after applying the adequate treatment, it is the rarest thing and in nearly all the cases it is noted in the journal, that both eyes were attacked at the first consultation. There has thus, in the great majority of cases, been an affection of the cornea—at any rate in one eye—during several days, before the patient was submitted to medical treatment. That is why we find the numerous cases of complete or partly blindness.

Among the 596 eyes of the surviving 298 children, no less than 496 (83%) were attacked, and of these 226 (46%) got blind. But among the mortal cases no less than 75 had both eyes attacked, and in 9 cases the right and in 4 cases the left was attacked before death occurred.

The necrosis of the cornea that appears with the keratomalacia spreads rapidly in depth thru all the strata of the cornea, so that perforation very soon occurs. The individual cards of registration note in 100 cases perforation in both eyes, in 68 in the right and in 49 in the left eye, in all 317 of the 659 eyes attacked (48%). In 186 cases the moment when the perforation occurred had been noted. Of these, in 127 eyes the perforation had occurred before the patient was submitted to treatment. In the first week after the disease had been established, perforation was observed in 45, in the second week in 8 eyes. After the second week only 6 perforations were observed. Spontaneous escape of the lens has been observed in 7 cases and hypopyon in 11 cases.

THE TREATMENT.

The idiopathic hemeralopia has been know since remote antiquity in nearly all countries of the world, and as a home remedy the treatment with liver has been applied—so to speak succesfully everywhere in olden days. In one of the most ancient medical works existing, the socalled "Eber's Papyrus," dating probably from about 1500 B. C., the liver therapeutics is recommended as a remedy against hemeralopia (Baron Oefele[71]). Also Hippocrates recommends "raw ox-liver dipped in honey once or twice, as much as he is able." In Japan and in China, according to Ishihara[116], the extract of hen's liver in honey and the liver of the hen and of the sheep were very much applied in olden days against hemeralopia. Later numerous physicians (among others Kubli[31], Schepotjew[46], Schtschepaatjew[53], Trantas[72], Bylsma[74], Romary[79], Lundsgaard[81], Alexander[85], Fabry[88], v. Sicherer[90] and Pincus[112]) have successfully applied raw, boiled or fried liver against hemeralopia and xerosis of the conjunctiva. Also cod liver oil has been used to a great extent against this disease. According to Romary[79], Baizeau, a French army surgeon, was the first who, in 1861, applied cod liver oil to soldiers against epidemic hemeralopia, with

good results. Later it has been applied successfully against hemeralopia and conjunctival xerosis among others by Gama Lobo[7], Weis[16], Snell[19], de Gouvea[23], Mori[82], v. Hippel[114] and Ishihara[116].

At first the medical science stood quite defenseless against keratomalacia. Most of the authors of those days recommended "good nourishment," without further designation of particulars, and the results were not favorable. Still several authors have, in the course of time, observed good effects from the treatment with milk. Milk is thus recommended by de Gouvea[23], Uhthoff[39], Baer[68], Hamburger[75], Romahn[105], Kapuscinski[108], Pincus[112] and Ishihara[116]. There was generally no question of a local treatment of the affection, beyond cleansing of the eyes, atropin and eserin (Thalberg[25]). Hirschberg and his pupils Kuthe[59] and Fehr[77] recommend for preventing the drying up of the cornea and the invasion of the bacillus a moist dressing fastened with collodion.

With our knowledge of the fat-soluble A we now understand the efficacy of the food-stuffs like liver, cod liver oil, animal fats, eggs and milk; but already a long time before we knew anything about the vitamines, both oculists and pediatrists in Denmark have known the excellent effects of the treatment with whole milk in cases of keratomalacia, and had applied this treatment to a great extent. Especially thru the clinical teaching in Havnegade and thru the works of Edmund Jensen and Bloch this treatment was generalized and has surely saved a good number of children from death, or from the hard lot of being blind.

The information of the reports about the treatment concerning this material is unfortunately rather scarce; but in nearly all the cases where the treatment is mentioned it is seen to have been based upon milk—most frequently raw milk—and in a number of cases also cod liver oil. Only in three cases no food rich in fat-soluble A has been applied as a treatment. In all these three cases the child died. In about 30 cases where I have had myself the opportunity of following the treatment, whole milk has been applied in all cases. In many cases the milk was raw, but sometimes it had been boiled for a short time, and there is nothing to show that the course of recovery was worse in these cases than in those where raw milk had been given. In numerous cases cod liver oil had been given, and the efficacy of this remedy was markedly good. Cod liver oil, according to the latest investigations, is one of the food-stuffs most rich in fat-soluble A. In most of the cases local instillations with atropin and bathing of the eyes with boiled water had been applied as well as, in a number of cases, sterile vaseline was instilled in the conjunctival sac; but generally we have the impression, that the local treatment is of less importance in the cure than the treatment with the fat-soluble A. In a number of cases it was necessary to do exenteration on account of panophthalmitis, and in several cases an optical iridectomy has secured partial sight of an eye affected with leucoma.

KERATOMALACIA IN ADULTS.

As already said, the keratomalacia in adults is rather infrequent. The literature, however, quotes a number of cases, but it is always pointed out that they are most i n f r e q u e n t. Thus Hildreth[12] mentions, already in 1870, a case of "Hornhautverschwärung" in a woman 52 years of age, who during 3 months had suffered from chronic diarrhea and who therefore was very emaciated and reduced. Shortly before she died she was attacked with necrosis of both corneæ, which were anesthetic. Xerosis conjunctivae is not mentioned. Later Schoeler[32] quotes 2 cases of sores of the cornea in adult patients, who both had been under an energetic treatment for reducing weight. There was no xerosis, but in one case it is indicated that the conjunctiva was lusterless, dry and folded on itself by the movements of the eye, so that it most probably had been keratomalacia or, as Schoeler calls the d i s e a s e, xerotic corneal disease.

In 1896 Ewmenijew[32] observed typical keratomalacia during a crop failure in Russia, when there appeared a great number of cases of scurvy together with hemeralopia and conjunctival xerosis, accompanied not infre-

quently by keratitis in the center of the cornea, which in the course of 4 to 6 days was completely destroyed. The disease attacked principally meagre and emaciated individuals. In the discussion at a conference about this question, Przybylski states that he has observed epidemic hemeralopia with necrosis corneæ in soldiers living in humid rooms of a fortress. Similar cases are said to have been observed by Schiele[69]. Bernhardt[73] quotes a case of keratomalacia in a drunkard 35 years of age who drank excessively and ate nothing. Uhthoff[84] observed among the inmates in a workhouse 2 cases of undoubted keratomalacia in adults, and was of the opinion that the cause was bad and insufficient food. Uhthoff quotes, in order to illustrate how seldom this disease is observed in adults, that among 120,000 patients affected with diseases of the eyes, he only observed 3 cases of keratomalacia. Flemming[95] mentions 2 cases of keratomalacia in adult inmates of a workhouse where the food was typically poor in fat-soluble A. Wicherkiewicz[102] observed the disease in a 31-year-old weaver, who had been a vegetarian for 10 years, and finally Saunte[145], in 1917, quotes a case of keratomalacia in a woman who, being toothless, lived upon beer-caudle, coffee and rolls soaked in milk, and who was restored to health by taking cod liver oil. To these may be added some cases of keratomalacia in adult patients affected with liver complaints. Hori[50] and Elschnig[62].

On reviewing the literature about xerophthalmia, we observe how frequently the disease appears in prisons, workhouses, educational establishments and the like, where the food rather often is undiversified and in former days even insufficient. In private homes where the food is more diversified, and where the boarders may often themselves decide on the composition of the food, the possibility that it should for a longer time be poor in fat-soluble A, is much smaller than in the above mentioned public establishments, where the food is ordered by the superiors according to a scheme that has to be followed strictly, and where there is no possibility of making up deficiencies by means of extra meals, etc. Only in the poorest homes and during crop failures the economical circumstances are so bad, that the food can for a long time be deficient in the more expensive food-stuffs which contain the fat-soluble A. Finally a hard régime can be so poor in fat-soluble A, that, if it is going on for a long time, it may involve the danger of the xerophthalmia. Likewise, on rare occasions, diseases of the digestive canal—especially if they impede the resorption of fats—may provoke xerophthalmia in adults.

When keratomalacia is observed in adults with so much less frequency than in children, it is due principally to the fact that the adult person is able to live a much longer time on food poor in fat-soluble A, without being attacked with keratomalacia. But there can be no doubt that the adult will instinctively look for nourishment containing fat-soluble A, if he needs it. This is the reason why keratomalacia as a rule only appears in adults in cases where the individual is prevented from following this instinct.

In conformity with this theory the cases of keratomalacia observed in adults included in this material all, except one single case, come from two asylums for imbeciles, where the disease had the character of an epidemic during some of the spring months.

The severest epidemic occurred in the asylum for imbeciles "A" during the months from March till June, 1917. Among about 450 inmates of the asylum, 53 were attacked with xerosis of the conjunctiva, and among these 13 got keratomalacia. The affected persons were from 17 to 72 years old. Five died (2 from bronchopneumonia and 2 from bronchitis with heart degeneration), 1 got blind in both eyes, 2 got blind in one eye and 1 was attacked with leucoma of one eye. The rest were restored to health, without any reduction of the sight. The majority of cases appeared in the beginning of May, the last case the 15th of June. There had not previously been observed xerophthalmia in the asylum. All the cases appeared in inmates; none of the attendants who got their food in the asylum were attacked with

the xerophthalmia. It was also observed, that the inmates could not keep their weight that year, which was previously extremely good; but the loss of weight was not especially marked in the inmates who were attacked with the disease of the eyes. The sanitary condition of the inmates in general was considerably lowered in 1917, especially during the first half, and the number of deaths was in that year more than double the ordinary number.

As far as the food is concerned the information says that only skim milk was used, and that eggs and cream were only given after the doctor's special prescription. Liver was not eaten in the asylum in the spring 1917. For sandwiches, for cooking and baking only vegetable margarine was employed. The same food had been given to the inmates also during the preceding year. The attendants, on the contrary, had butter instead of margarine, but otherwise ate the same food as the inmates.

As it will be seen, the food of the inmates had been poor in fat-soluble A. The fact that the disease did not appear until 1917, is due probably to the fact that the quantities of fat-soluble A of the food had not been very much under the minimum of fat-soluble A, which is necessary for keeping adult persons free from the keratomalacia, for which reason the reserve of fat-soluble A had been slowly exhausted; so that rather a long time elapsed, until it was completely exhausted (in 1917) among some of the inmates, and the keratomalacia broke out. The small content of fat-soluble A of the skim milk in the spring 1917 may also have some part in the fact, that the epidemic broke out just at that moment. The attendants were not affected with the keratomalacia, because they had the whole time eaten butter. Since the spring 1917 all the persons in the asylum have had butter, and since then no case of xerophthalmia has been observed there.

Another lesser "epidemic" occurred in the spring 1920 in the asylum for imbeciles "B," where about a hundred imbecile men are maintained. Five cases of keratomalacia appeared among these in the month of May, which I myself had the opportunity of observing and following. All the cases presented the typical symptoms of keratomalacia and xerosis of the conjunctiva. The ages of the patients were 20, 23, 24, 35 and 50 years. In all the cases only one eye was attacked, when the disease was established; and as the treatment was immediately initiated, the other eye was not attacked. On examining all the other inmates of the asylum, nobody was found attacked with xerosis conjunctivae. The food in this asylum consisted during the spring of 1920 of: Breakfast, barley-gruel with separator skim milk or beer, black bread with vegetable margarine and skim milk cheese. Dinner: milk-food of separator skim milk, sweet gruel, beer-caudle, cabbage, meat four times a week, fish (mostly klip-fish or pickled herring) once a week and potatoes. Supper, as breakfast. Coffee with cream and buns only on Sundays.

While during the previous years a little butter had been employed now and then, the inmates got exclusively vegetable margarine after the first of October, 1919. It will thus be seen, that the food was in the beginning of 1920 extremely poor in fat-soluble A. Among the food-stuffs containing fat-soluble A only the tiny bit of cream taken with the coffee on Sundays, and the fish in those weeks where the fish was fat (in the conserved salt herring the fat-soluble A is, however, most probably destroyed by oxidation) may have had some importance for supplying fat-soluble A. It will easily be understood that even adult persons living upon a food so poor in fat-soluble A may be attacked with keratomalacia. It is only to be wondered that not many of the other inmates showed symptoms of incipient xerophthalmia.

The inmates were weighed every month, and figure 13 shows 2 curves illustrating the average weight; the lower curve shows the average weight of the 5 inmates who were seized with keratomalacia, and the upper curve shows that of the rest of the inmates. While the latter did not show any visible loss of weight during the spring months of 1920, there is a considerable loss of weight to be noticed among

the inmates seized with keratomalacia. These were treated with whole milk and cod liver oil (besides local treatment), and also the rest of the inmates had whole milk prescribed. The curve illustrates how the average weight, shortly after the treatment had been initiated, increased rapidly. The affection of the cornea was also cured rather soon. One eye got staphylomatous, one was cured without reduction of the sight, and the rest of the eyes healed up with smaller or larger leucomas.

As will be seen, the epidemic way in which the cases here mentioned appeared may be explained from the uniform alimentary conditions of the affected persons; and as it was seen, the food in the cases quoted had been so poor in fat-soluble A, that the appearance of the keratomalacia and the facts of the same, may very well be explained from the theory that the xerophthalmia was due to the lack of fat-soluble A. Moreover, these "epidemics" give a serious reminder of the attention that ought to be paid to the planning of an alimentary scheme for the greater establishments of this kind, and it should especially be pointed out, that the food, besides containing the sufficient number of calories, etc., should be in a sufficient degree rich in fat-soluble A.

Besides the cases of keratomalacia here mentioned, our material includes only one case of this kind, in a man 59 years of age, living in a town in Jutland. In September, 1920, the patient was seized with a strongly marked xerosis of the conjunctiva in both eyes, and in the right eye a lighter attack of keratomalacia, which in the course of 3 weeks healed thru the treatment with cod liver oil. We have no information about the food of this patient.

XEROSIS CONJUNCTIVAE.

Besides the 453 cases of keratomalacia there were gathered 148 cases of xerosis conjunctivæ without affections of the cornea; of these, 133 cases appeared in children and 15 in adults (over 16 years). These cases were seen to pass off in all respects just as we from our experience of the severe cases of keratomalacia might expect these milder cases to do.

As it will be seen from the above treatise on the nutritional conditions in Denmark, during the years 1909-1920, and the appearance of the xerophthalmia in Denmark during the same years, the whole matter appears as a great experiment which surely is unique, as to the importance of the vitamins, especially fat-soluble A, for the nutritional conditions of a whole nation. The xerophthalmia is the manifest symptom of the lack of fat-soluble A, which appears as a constitutional disease for which I propose the name: dystrophia xerophthalmica. But the latent avitaminosis, due to the lack of fat-soluble A, which amongst other things manifests itself thru a great susceptibility and lowered resistance to all infections, has, undoubtedly, been present in a far greater number of cases than here mentioned. Therefore, not only for the developing of the xerophthalmia, but also for many other diseases these nutritional conditions have surely been of great importance.

LITERATURE.

The following abbreviations are used:

A. f. A.	Archiv für Augenheilkunde.
A. f. O.	Archiv für Ophthalmologie.
B. kl. W.	Berliner klinische Wochenschrift.
Bioch. J.	Biochemical Journal.
D. m. W.	Deutsche med. Wochenschrift.
Diss.	Inaugural-Dissertation.
Hosp.	Hospitalstidende.
J. d. O.	Jahresbericht der Ophthalmologie.
J. B. Ch.	Journal of Biological Chemistry.
Kl. M. f. A.	Klinische Monatsblätter für Augenheilkunde.
M. m. W.	Münchener med. Wochenschrift.
Tr. Oph. Soc.	Transactions of the Ophthalmological Society of the United Kingdom.
U. f. L.	Ugeskrift for Læger.
W. f. Th. u. H.	Wochenschrift für Therapie und Hygiene des Auges.
W. kl. W.	Wiener klinische Wochenschrift.
W. m. W.	Wiener med. Wochenschrift.

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REFORM DIET AS A THERAPEUTIC MEASURE IN OPHTHALMIC PRACTICE.

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The importance of toxemia of gastrointestinal origin, along with focal infections, as a cause of ocular disease is here emphasized. It is held that heavy starches to be digested in the intestines should not be mixed with proteins, that must be digested in the stomach; and the excessive use of sugar is condemned. The influence of diet in etiology and the therapeutic value of its correction, is illustrated with reference to corneal ulcer, keratomalacia, headache, glaucoma, choroiditis, and other ocular conditions. Read before the Medical Society of New Jersey, June 22, 1923.

In presenting a paper on "Reform Diet," I wish it to be understood that it is part of my remedy for the "three Ts." The ramifications of the "three Ts" (teeth, tonsils and toxemias of the intestinal tract) are so interwoven, that to my mind it is impossible to dissociate them. In thinking of any one of the "three Ts" in connection with a patient, the other two invariably come into my mind. They go hand in hand.

We know that gastrointestinal disturbances and severe septic intoxication, are at times brought on, or aggravated, by oral sepsis, diseased tonsils, or wrong food combinations, and not infrequently by all three. I said at the A. M. A. meeting in 1919[1], that oral sepsis was one of the greatest menaces to the human race! I reaffirm that statement. I also said that negative evidence does not exclude the possibility of infection from the tonsil, and that bacteriologic examination of all removed tonsils which I have examined, demonstrated the presence of organisms. I have been driven to the conclusion that a healthy tonsil is a scarce article. There is no doubt in my mind that the constant swallowing of infected secretions from the mouth and tonsils, is one of our greatest sources of danger. Septic infection may be absorbed into the circulation from both, and thus diseased teeth and tonsils in two ways become enemies of the host and gnaw at the vitals.

E. Libman,[2] in a paper on Acute Bacterial Endocarditis said: "The important thing is to systematically rid all patients having valvular disease, of the ascertainable foci of infection," (teeth, tonsils, etc.), and he further adds that the question of prevention seems to be the most pressing.

Behan,[3] said:—"We see continually in our private dispensary and hospital work, all the changes characteristic of albuminuric neuroretinitis in varying combinations. In the absence of nephritis we do not hesitate to ascribe as a cause for these changes the "three Ts" of Bell, the anemias and infectious diseases." Olmstead[4] said: "I feel safe in saying at this time, excepting trauma and venereal disease, we can, or will eventually, prove every diseased condition of the eye due to some one of the three recognized sources of focal infections, viz: "the three Ts." In the light of all this, what is the sense and what is the use of putting patients on a diet, unless you are going to get rid of infected teeth and diseased tonsils? We must attend to the hygiene of the mouth and throat.

We come now to my "Reform Diet." I hold and believe that diet is common sense applied to living. All must agree to that, if we are to make any real progress in overcoming toxemia. We must consider the fuel that we put into the laboratory that nature maintains in the stomach. The day is at hand when the progressive ophthalmologist must take up with his patients the question of diet. How many oculists warn their patients against excessive consumption of sugars, sweets and starches, and wrong food combinations. Many ophthalmologists are so occupied with the purely ophthalmologic aspect of a case, that they pay absolutely no attention to the practical important etiologic factors.

When we realize that ninety out of one hundred persons die from some secondary disease, the result of some focal infection, it is high time that we were studying the causes of focal infections. McCollum[5] comes out in his

latest book on "The Newer Knowledge of Nutrition" and says, that no one will be likely to dispute the statement that few people are entirely well, and that their failures, indiscretions, fits of temper, tendency to complain, their lack of enthusiasm, their inefficiencies as creatures, are in a great measure the result of ill health, and he adds that there can be no reasonable doubt that the diet may deviate from the optimal even in the United States, to a degree which can easily cause the results we see.

Now if what McCollum says be true, and I believe it is, and if we have been living on a socalled well balanced diet which includes meat, bread, vegetables and sweets, and also meat, potatoes, vegetables and sweets, and heavy starches and proteins at the same meal, it is high time we were making a change, and that we tried some other form of diet, and thus endeavored to prevent the factors concerned in the production of this formidable array of ills, of which McCollum speaks. Hence my reason for presenting to you for consideration my "Reform Diet," as a therapeutic measure in ophthalmic practice.

Let us keep a few things clearly in mind. Heavy starches and proteins must not be eaten together at the same meal; for the reason that starches are digested in the mouth and intestines, and proteins in the stomach. What is the sense of eating foods together that fight in the stomach? Why not eat them separately, and at different meals?

When meat is eaten it causes a flow of gastric juice. The elements of a food that are peculiarly fitted to a certain digestive fluid, cause this fluid to be secreted—so that when meat and highly starchy foods are eaten together, the meat will be digested in the stomach, but not the starch. Now all the time the starch is in the stomach, it is not only not permitted to be digested, but it also hinders the digestion of the protein or meat. So we get an acid fermentation in the stomach from the starchy foods with the formation of volatile and nonvolatile acids and sometimes marsh gas, hydrogen and alcohol. It is the acid fermentation produced by these wrong food combinations which in the end leads to toxemia.

Starchy foods, of course, we need for energy and heat, but they must be eaten with nonstarchy vegetables, salads and fruits, in order that they may all pass out of the stomach at the same time, and in that way we are able to prevent acid dyspepsia and acid fermentation in the stomach.

Matthews[6] in the third edition of his "Physiological Chemistry" says that starches, or carbohydrates, are digested by the ptyalin of the saliva, the amylopsin, maltase and lactase of the pancreas, and the invertin of the intestinal juices, so that they are all reduced to a state of monosaccharids. He further adds that some of the carbohydrates are further broken up by the bacteria with the resultant formation of lactic acid, alcohol, marsh-gas, hydrogen and butyric acid.

Sherman[7] adds that among cases of excessive bacterial decomposition in the digestive tract, the fermentation of carbohydrates with the production of organic acids, and possibly alcohol, is most likely to occur in the stomach; while putrefaction of proteins occurs mainly in the large intestines. Then if what Sherman and Matthews say be true, why not eat carbohydrates at a time when they won't be detained in the stomach? Sir Francis Hare[8] comes out flat-footed, and says (in "Food Factor in Disease," Vol. I) that there is nothing in the stomach to digest the starches. J. H. Tilden[9] says the same thing and has advocated for a long time this diet of not mixing starches and proteins at the same meal. Deeks[10] also has pointed out that there is nothing in the stomach to digest the sugars.

I am a firm believer in the baneful effect of sugar as a systemic poison in diseased conditions of the eyes. We all have seen the baneful effect of sugar as a systemic poison in diseased conditions of the eyes. We all have seen the baneful effect of candy and sweets in children coming to us with

eczematous conjunctivitis. Stay away from sugars and sweets and get the necessary sweets by eating fresh fruits and vegetables. One hundred years ago the total per capita consumption of sugar in the United States was less than nine pounds per annum. Since then it has increased by leaps and bounds, until now this country is probably the largest consumer of sugar in the world. Kellogg and Taylor estimated the per capita use of sugar just before the war, at close to four ounces a day per individual. Estimates of sugar in the blood are of value in the treatment of all cases of obscure eye conditions. Chace said in 1920 at the annual meeting of the Medical Society of the State of New York, that sugar is perhaps responsible for producing more hyperacidity than any other single article of diet, and that the great increase in the prevalence of hyperacidity during the last decade is largely due to increased sugar consumption.

There is a growing demand for a scientific supervision of the diet in health and disease, and an increasing realization of its importance. Hence my great interest in "dietary reforms." I want to emphasize the importance of using certified raw milk, and of eating raw fruits, green vegetables and salads. Again, certain important substances occur in animal fats which should form a part of the diet, such as fats from meats, butter and cream. We should consume at least one quart of milk or its equivalent in dairy products every day, and raw food and raw fruits should be eaten as salads and desserts once or twice daily to insure the required amount of vitamins. It seems that in the course of cooking or drying s o m e t h i n g essential is destroyed. We ought every day to eat freely of some leafy vegetable food such as spinach, cabbage, beet tops brussels sprouts, lettuce, dandelions, pepper-grass and Swiss chard. They have unique properties. The consumption of these leafy vegetable foods will go far towards overcoming constipation.

I have so arranged the "reform diet" that milk can be taken at the proper time. I am a firm believer in the food value of milk, the most desirable single article of food for infants and children, and being more and more appreciated by adults. Funk was the first to suggest the relationship between the composition of the milk, and some deficiency diseases in children. Funk stated that on heating milk certain vitamins become more or less completely destroyed. Therefore great advantage lay in a certified raw product, safeguarded from contagious diseases and containing a more or less constant fat content. Milk taken with the ordinary meals f r e q u e n t l y causes catarrh, because of overeating and overloading the stomach. Milk must be taken with a starch or fruit, and not with a full meal, because one quart of certified raw milk is a meal in itself. A hearty eater, who drank about two glasses of raw milk with each meal, three times daily, developed a catarrhal state, followed by intestinal toxemia and keratitis of both eyes. I found his tongue heavily coated, his breath very foul and his urine loaded with indican, also he was a sufferer from constipation. His teeth were apparently healthy. I was able to extrude pus from his tonsils.

Examination of his eyes showed Mooren's ulcer of right eye and incipient ulcer of the left. The ulcer in the right eye had developed from the margin of the cornea above, and had extended all the way a r o u n d the cornea. The gray, cloudy margin of the ulcer was undermined and could be elevated. Moreover, there were marked inflammatory complications. He had been treated for six weeks by an ophthalmologist. This oculist had cauterized the ulcer with carbolic acid several times and had used the actual cautery, also hot fomentations and atropin were being used. All I did for him was to have his tonsils enucleated at once, put him on my "reform diet" with hot applications to his eyes—bichlorid vaseline—1-5000 in both eyes night and morning, and atropin solution in his eyes occasionally; while his colon was thoroly emptied every day by means of an enema of water

at blood heat. This patient made a good and rapid recovery, and returned to his native state in two weeks, where he continued the treatment for four weeks more. Doctor H. A. Wakefield of Charlotte, North Carolina, in a letter recently, said that Mr. B. now had normal vision in both eyes.

RULE OF THE REFORM DIET.

The principle of the reform diet is simple and very plain. Not to mix heavy starches and proteins at the same meal.

Heavy starches—Bread, whole wheat, white corn or rye, and crackers. Potatoes, rice, macaroni, corn, lima beans, dry beans, and also sugar.

Heavy proteins—Meats of all kinds, fish, shell fish, chicken, duck, turkey, eggs and cheese.

I have arranged a "Diet Sheet" but one may make his own, if he does not violate the rule and principle. This system of diet, which I can strongly recommend, is in no respect a radical one. It includes the best features of several types of diet. Eat any kind of food you like or choose, provided you eat it at the right time.

McCollum[11] says that in the last century changes in the habits of the population of Europe and America have brought about changes in diet. The changes have been in favor of highly milled cereal products, muscle meats, potatoes and sugar. *This diet is a failure* in animal experimentation and is proving a failure in human experience. Evidence that this is true is abundantly supplied in the percentage of underweight children in our schools and in the prevalence of rickets, small jaws, and teeth which are irregular, poorly developed and which decay easily. Personally, I am convinced that there is something wrong with our way of living. McCollum has proved it with animals, and my experience with this reform diet over a period of years, in my clinics and with my private patients, coincides exactly with his findings. It is a never ending fermentation in the stomach and putrefactive condition in the intestines, brought about by wrong food combinations, bad teeth, and diseased tonsils, that is destroying the human race. We must not lose sight of the fact that toxemia ranges in intensity from an ordinary cold to severe acidosis. And also do not forget that we can have excessive fermentation as well as excessive putrefaction in the same individual and at one and the same time. Sherman[7] adds, that from eating large quantities of meat and sugar together, a type of fermentation ensues in which oxalic acid is produced and which must be highly injurious and most distinctly toxic. Also Lieb[12] has proven that oxalic acid is produced in the colon by the bacterial decomposition of sugars, and that oxalic acid produces definite toxic symptoms even in small amounts.

KERATOMALACIA.

Extensive use of faulty foods which are low in vitamins, prevails largely among many civilized people at the present time. McCollum and Simmonds have proved keratomalacia to be due specifically to lack of fat soluble A vitamin in the diet of animals. Bloch[13] has observed 40 cases of keratomalacia among children fed on highly centrifuged milk, fat being given in the form of vegetable margarine. A cure was obtained in most cases with the aid of raw milk, or cod liver oil. Bloch believes that this form of ophthalmia occurs quite frequently in Denmark and may often be a cause of blindness.

McCarrison[14] states that this disease occurs not infrequently in India, from a diet of polished rice and vegetable oils, and is curable with cod liver oil. I have observed only five cases of keratomalacia, all five in young children. The diagnosis is easy: all of them were suffering from severe malnutrition. The skin was dry, shriveled and scaly. Raw milk and cream, with the addition of cod liver oil, effected a cure in three of the cases.

HEADACHES AND DIZZINESS DUE TO FOOD INTOXICATION.

The following is a most interesting case and was unquestionably due to

"wrong food combinations." This patient, Dr. G. F. R., consulted me for occipital and frontal headaches, which came on from time to time. He also complained of great dizziness without loss of appetite. In fact, he said to me: "I am so dizzy that I can't properly do my work." Upon examing his eyes I found that his glasses were correct. Looking him over for the "three Ts" I found his teeth excellent, his tongue coated, and his tonsils had been removed. Then I took up with him the question of diet, and this is what he was eating every day.

Breakfast—Orange juice, cooked fruit with sugar and cream, eggs and bacon, or fish, coffee, toast or muffins.

11:30 A. M. Egg-nog.

Luncheon—1 P. M. A big bowl of thick soup, (meat) with lentils, beans and peas, black bread and butter.

4:30 P. M.—Arrow root biscuit and two cups of tea.

Dinner—7 P. M. Soup, meat, potatoes or rice, or macaroni with cheese, fresh vegetables, salad with French dressing, and a sweet dessert.

He was especially fond of puddings. The diagnosis was easy in this case. I at once told him that he was suffering from intestinal toxemia due to wrong food combinations—eating heavy starches and proteins at the same meal, and overloading the stomach. He is a very busy dentist and thought the more he ate, the better he could work. In other words he was catering to his appetite. I put him on "reform diet," which he has been following now for twelve months. His dizziness has entirely disappeared, and his headaches have been completely relieved. He says that he feels like another man, and works from 8:30 A. M. to 6 P. M. six days a week.

GLAUCOMA.

We come now to chronic congestive and noncongestive glaucoma, the cause of which is little understood. Glaucoma is not merely a local disease. It is the outcome of prolonged chronic autotoxemia, provided we can exclude syphilis; and we may have both infections in the same case. Glaucoma cases must first have the Wassermann test. A vitiated blood supply not only acts deleteriously upon the nervous apparatus, but coincidently with this, gives rise to inefficiency of the secretion of every endocrin gland in the body, the natural consequence being an absence of healthy cell metabolism, and the dire disturbance which must follow. Chronic constipation, gout, rheumatism, arteriosclerosis or uricacidemia, or all of them exist in those who become the victims of chronic glaucoma. Professor E. Fuchs[15] says, that the relation between glaucoma and the thyroid is supposed to be due to the influence of the gland on the salt metabolism. In glaucoma cases, the salt in the blood amounted to 0.72% as against 1.13% in healthy persons. Imre got good results, according to Fuchs, by thyroid treatment in glaucoma cases, and he found the tension lower during pregnancy when the thyroid becomes enlarged. I said at the New York Academy of Medicine, five years ago, that fewer of our glaucoma cases would come to the operating table, if we would only remove their focal infections and the profound toxemia from which they all suffer. Lagrange comes out in his latest book and lays stress on a point that is often forgotten, namely that glaucoma is generally not merely a local disease, but an ocular disease occurring in an already diseased organism. Elliot, also, in his latest book on Glaucoma says: "It seems probable that one of the greatest advances of the future in the treatment of glaucoma may lie in the direction of the early and scientific combating of the source of autointoxication."

My conviction is that glaucoma must be treated constitutionally, as well as locally or surgically. I have the records of 44 cases of glaucoma which I am treating according to my method; and so far not one of these twenty-two cases has come to the operating table. One of these cases has been under observation for nearly ten years. In all my cases I use nitrat of pilocarpin. The eye stands it better over a period of years, and I think

it is less irritating. Posey first called my attention to it, and as Posey says, the drug must be used constantly and continuously every day at least three times.

The history of this patient whom I have had for ten years is very interesting. At the time I first saw her, she was 70 years of age. She complained of disturbed vision, as tho looking thru a fog, and the street lamps had a halo of colored rings. She had recently been in the hands of two oculists, both of whom had made a diagnosis of chronic glaucoma. Both oculists wanted to operate on her. When I first saw her, she had a distinct glaucomatous disc in each eye; her anterior chamber was very shallow, with the pupils moderately dilated. Schiötz recorded 35 m.m. of Hg. in each eye. Her blood pressure was 180. Her urine was loaded with indican. Upon looking her over for the "three Ts" I found that her tongue was heavily coated, her breath foul, and she suffered from constipation. X-ray of teeth was negative. She gave a history of consuming considerable sweets, and was in the habit of eating meat twice daily and taking a great deal of coffee.

I restricted the use of the eyes for close work, and put the patient on a diet. The exclusion of heavy meats and stimulants, such as tea, coffee, alcohol and tobacco is most essential in order to obviate the final need of surgery in these glaucoma cases. E. Lyman Fisk[16] says that it is a poor compliment to the human race to say that life is unlivable without alcohol, tobacco, coffee and tea. Also with this patient I used pilocarpin nitrat, one per cent three times daily, hot bathing for the eyes three times daily, and a hot bath at bed time. I also gave her a vial of bacillus bulgaricus three times daily after meals, and careful attention was given to evacuation of the bowels every day. In addition to this the colon was thoroly emptied once a week by an enema of water at blood heat. This regimen has been kept up for ten years, with practically no loss of vision and only a slight impairment of the fields. Is not such a method as this far preferable to an operation on a patient over 70 years of age?

Let me give one more example of chronic simple glaucoma which is most interesting. The patient, also a woman, age 45, was operated upon for chronic glaucoma by one of our well known surgeons. When I first saw her three years ago, I looked at her eyes and saw that she had had a proper operation performed, but the vision remained very poor. She came to me to be treated for headaches, and pain in her good eye. I found the anterior chamber very shallow. The tension was up. Upon looking her over for the "three Ts" the X-ray of the teeth revealed abscesses at the roots of eight of her teeth. There is nothing that can destroy bacterial life in the pus sacs at the roots of infected teeth except extraction, so we had her infected teeth removed. Her tonsils had already been removed. Her tongue was heavily coated, her breath was foul and she was suffering from constipation. Moreover, her urine was loaded with indican. Her Wassermann was negative. I placed her on the "reform diet" and the usual treatment for glaucomatous eyes. I gave her Bacillus acidophilus. We know that Bacillus acidophilus is the protective organism of the intestinal tract. Eggston and Norman[17] have proved that beyond question. I give the B. A. for one month at a time, and then I give small doses of iodin for one month at a time to maintain the endocrin balance.

This *iodin treatment,* which was first brought to my attention by A. J. Quimby,[18] is so valuable that I give the formula.

℞ Iodin crystals.....Gr XXX
Ammonia iodid.....Gr XX
Alcohol .....ʒ IV
Glycerin .....q. s. ʒ I
Misce et signa: one drop in glassful of water once daily.

For patients who have been travelling the road of toxemia for years, and where the toxins have been accumulating in the blood, small doses of iodin are exceedingly valuable. As the

result of this treatment this patient has normal vision and normal fields after three years. No effort had been made in this case to improve the quality of the blood stream, until she came under my observation.

Of course I operate for glaucoma when I find it is necessary—that is when the treatment which I have outlined is not effective. This, however, is rarely the case. I had a chemical examination of the blood made in six of these glaucoma cases. In four of the six cases, the blood chemistry showed uric acid content above normal. The uric acid ranged from 4.5 to 6 mg. per 100 cc. of blood. According to De Niord and Bixby, when the blood chemistry reveals uricacidemia, that is enough to justify a diagnostic finding, indicating a chronic infection somewhere in the body. I maintain that in all chronic simple glaucomas, we should insist upon a chemical blood test for uric acid.

This general routine treatment which I advocate in glaucoma cases is also carried out by me in all diseases of the eye, such as episcleritis and deep scleritis, retinitis, optic neuritis, eczema of the lids, herpes febrilis, herpes zoster, edema of the lids, toxic amblyopia, immature cataracts, both hard and soft, choroiditis, keratitis of all forms, chronic conjunctivitis and blepharitis, iridocyclitis, plastic iritis, chronic uveitis, recurrent hemorrhages in the vitreous body, sympathetic ophthalmia, migraine, angioneurotic edema and papilledema. Of course, in conjunction with this general routine treatment, the necessary local remedies for the eyes must be used. Salvarsan and inunctions of mercury and tuberculin are also added when indicated and necessary. In chronic conditions of the eye, where the tuberculin test is positive, I find myself giving less and less tuberculin, and relying more and more upon fresh air, sunshine, exercise, my "reform diet" and the iodin treatment, as well as close attention to the sanitary condition of the colon.

CHOROIDITIS.

Several well known oculists have recently reported cases of choroiditis, keratitis and episcleritis cured as a result of the diet. I have any number of such cases among my records. There is one case of exudative choroiditis in a young Federal Board student, aged 24, of which I would like to speak. History as follows:

The sight of O. S. was lost in France, as a result of shell explosion, O.D. vision 10/200. On examining his eye with the ophthalmoscope there was a large cloud of floating opacities in the vitreous, so thick that the details of the fundus could not be seen. I admitted him to the U.S.P.H. Hospital No. 38 (New York Polyclinic) where the Wassermann was negative. Looking him over for the "three T.s" I found that his teeth were in fine shape, but his tonsils were filled with pus.

I had his tonsils enucleated, and put him on the reform diet. Hot applications to the eyes for one hour at a time, three times daily, were given and also a weak solution of atropin was used. Careful attention was given daily to complete evacuation of the colon. The patient began to improve, and left the hospital at the end of two weeks with instructions in regard to diet and enemas. Six months later I saw this patient, and much to my surprise he had 20/20 vision. On examining the vitreous no floating opacities could be found, but several patches of choroiditis could be seen in the peripheral portion of the fundus.

CONCLUSIONS.

1. After five years experience with the "Reform Diet" I feel that I stand on a firm foundation in recommending it as a therapeutic measure in ophthalmic practice.

2. I agree with Meller, who says that sympathetic ophthalmia may be produced by endogenous infection, and he adds that the organisms in the blood settle in the tissues of the eye weakened by injury, the organisms becoming virulent and finally reaching the other eye thru the blood stream. I maintain that a house cleaning process is in order in every obscure condition of the eye.

3. Alimentary toxemia is beyond question responsible for a large percentage of diseases of the eye. The relation of food ingested to alimentary toxemia is very important. Most people eat far too much and might with advantage eat very much less. This idea should be constantly kept in mind by every ophthalmologist.

4. In acute and chronic diseases of the eye, would it not help matters materially if we adopted measures, such as a "Reform Diet", the systematic removal of ascertainable foci of infection and the closest attention to the sanitary condition of the colon, with a view to removing the toxic condition of the blood?

5. When a man like McCollum says that a diet composed of milled cereal products, muscle meats, potatoes and sugar is a failure in animal experimentation, and is also proving a failure in human experience, then I think it is high time that we "STOP—LOOK—and LISTEN."

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OSTEOMA OF THE ORBIT.

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This paper reviews some of the striking cases of orbital osteoma previously published, especially those found in recent American literature. It also reports a case in which the proptosis was scarcely noticed until orbital inflammation occurred. The tumor arose from the orbital plate of the ethmoid, part of which was removed with it. The eyeball was restored to normal position and retained full vision. Read before the American Ophthalmological Society, June 19, 1923.

Osteoma of the orbit has, according to Parsons[1], been recorded since 1506, when Veiga reported his experience. Since that time many observations have been made. Lediard described a case in which a 9½ ounce tumor protruded between the upper lid and eyebrow, causing a rupture of the globe and finally extrusion of a mass which measured 4½ x 5 inches. Hilton saw a 14¾ ounce osteoma which after destroying the eye by pressure was spontaneously expelled. A tumor which weighed 351 grains was seen by Stevenson. Cooper reported a bilateral orbital exostosis which seemed to come from the antrum, and which by extension into the cranial cavity produced sufficient vascular change to cause an apoplexy from which the patient died. Middlemore also referred to a large osteoma.

To attempt the complete report of literature would be time consuming and serve little good. Several citations have been made by members of this Society. The first case so presented was by Norris in 1884[2]. The growth springing from the floor of the right orbit produced optic atrophy with blindness. The eyeball and tumor were removed. Recovery was good.

In 1890 Pooley[3] removed a large osteoma and preserved the eyeball. This was in a girl of twenty-three years, in which the mass filled the upper, inner angle of the orbit. The eye projected outward and somewhat upward with marked limitation of motion especially in and down, with optic neuritis and vision of 20/200. An incision was made in the brow margin and caried along the side of the nose. The tumor was chiselled free, dislodged and on removal weighed 26 grammes. Reeve in discussion stated that he had removed an osteoma which came from the frontal sinus and the patient recovered with full vision.

Sattler[4] reported a case in 1896. His patient, a male twenty-five years old, had a tumor near the upper, inner margin of the right orbit which caused an enormous degree of exophthalmos, the globe being turned outward, downward and forward. The growth hard and nodular weighed 15.3 grammes. Recovery following operation was uneventful.

The year following Sattler's presentation, 1897, Norris[5] cited the case of a male of twenty-four years who had a tumor in the nasal wall of the right orbit, which existed for six years, causing severe headache with dizziness and diplopia but without other interference in vision. The osteoma removed by him weighed 258 grains.

In that same year Sattler supplemented his report of the year preceding.

In 1898 E. Fridenberg operated on a case in which from the center of the superior orbital margin a mass protruded pushing the eye backward and downward. When removed it measured 31 x 25 x 20 mm.

In 1903 Percy Fridenberg detailed his experience with a twenty-one year old man, in which the eye protruded and was dislocated outward and downward without, however, interference with globe motility. Incision was made along the orbital ridge below the brow; a tumor measuring 29 x 14 x 4 mm. and weighing 100 grains was removed. When the osteoma was freed there was a small amount of mucopus which welled from the ethmoid attachment. The patient made a good recovery.

In 1911, Sweet[8] gave an account of the case which had been recorded by Norris in 1897. The patient had an antrum infection and was operated with

cure. Veasey[9] in 1915 showed X-ray pictures and gave the clinical history of a case which, however, was not operated upon.

Herman Knapp[10] in the Archives of Ophthalmology for 1880 reported a cure and detailed the operation for the removal of a 36 x 29 x 24 mm. osteoma.

Andrews[11] in 1887 in a most comprehensive article reviewed the literature up to that time and reported two new cases.

Fig. 1. Exophthalmos and edema of lids shown October 22, 1922, from in front.

Jackson[12] in 1892 presented a report before the American Medical Association of the successful removal of a 13 gramme osteoma from the orbit with the preservation of vision.

Arnold Knapp[13] in the Archives of Ophthalmology for 1906 illustrated and reviewed two cases operated upon by him. In one the tumor measuring 2 x 3 cm. was located deep in the orbit, the other was 4 x 2 cm., weighing 5.15 grammes; in both recovery was complete.

Blake[14] reported a huge osteoma of the orbit. He stated that the tumor was the size of a closed fist and came from the left frontal sinus. He advised against operation. The patient was operated upon by a general surgeon and died on the table.

From this synopsis of the cases reported by members of this Society or before this Society, it is evident that experience with this type of lesion is not unusual. Lagrange[15] in 1904 collected 148 cases. The older writers seemed to have been cognizant of the condition and yet must have been fearful of the outcome, as is shown by the report of the very large growths, notably those by Lediard and Hilton. LeMond says that in 60% of these cases death follows operation. Lagrange, however, feels that 6% is nearer the present truth, altho Cumston considers the mortality to be 10%.

Because the disease presents certain curiosities in the form of development,

Fig. 2. Condition shown October 22, 1922 seen from left side.

in the length of time individual cases are under observation and in the end result, the following experience is reported in detail.

REPORT OF CASE.

E. L. age seventeen years was referred by Dr. Frank B. Wheeler of Hudson, N. Y., with a history that a week ago he had had some pain with headache both localized around the left side. Three days later there was redness of the left eyeball and lid conjunctiva, followed in a few hours by an increased prominence of the globe.

First examination made October 2, 1922; right eye pupil 4 mm. slightly oval but reacting promptly to light and accommodation. Cornea and media clear, disc distinctly outlined. No limitation of external motion.

The left eye showed an extreme edema and congestion of the entire orbital region with marked exophthal-

mos. The globe was 11 mm. more prominent and slightly lower than the right, with motion limited in all directions, practically abolished upward. There was a firm subperiosteal, nodular protuberance to the upper and inner side of the orbit. To the temporal side of the upper lid there was fluctuation. Pupil 5 mm., regular, active, media clear, disc distinct with venous engorgement. The diagnosis of osteoma with secondary orbital abscess was then made.

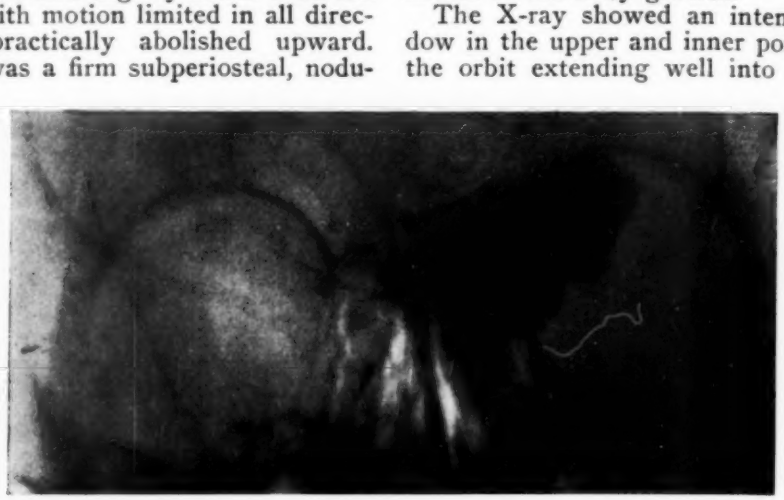

Fig. 3. Radiogram taken anteroposteriorly showing growth at upper inner angle of orbit.

Under ethyl chlorid general anesthesia, an incision was made thru the fluctuating area. There was a considerable amount of glairy mucopus with no change in the contour of nasal side of the orbit. Hot compresses caused the inflammatory symptoms to subside, and he was told to return when the lids were white, at which time an operation would be performed to attempt the removal of the bony growth.

The X-ray showed an intense shadow in the upper and inner portion of the orbit extending well into the orbital cavity and leaving but a thin layer of bone between it and the dura. The frontal sinuses were large, clear and easily outlined.

Nine days after draining the orbit, the left eye was 3 mm. more prominent than the right and remained markedly divergent as well as 5 mm. lower.

On October 26th, 1922, he was given ether and the skin cleaned but the eyebrow was not shaved. An incision was started in the outer third of the left eyebrow, carried across to the nose and then downward along the side of the nose, going thru the periosteum. All of the soft tissues were dissected with the periosteum uncovering a large,

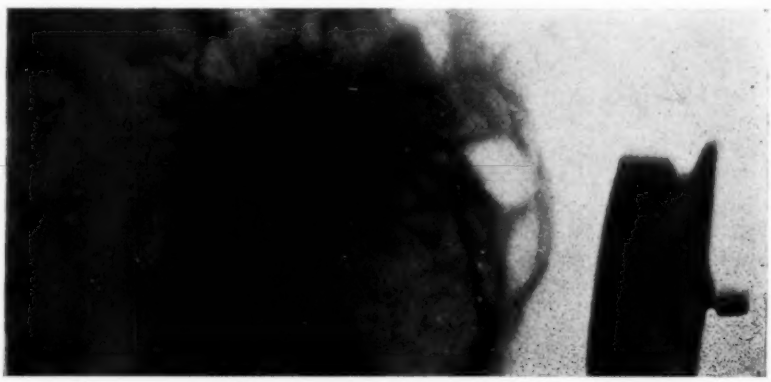

Fig. 4. Radiogram lateral exposure, shadow of growth in upper part of orbit opposite posterior ethmoid.

nodular mass and draining about fourteen drops of glairy, greenish-brown mucopus from the deepest point of the growth's attachment. The bone growth was so large, so irregular and so heavy Volsella forceps. The point of attachment was irregular in outline, its greatest diameter being 12 x 11 mm. There was on its surface an almost perfectly round channel, 2 mm. in diameter

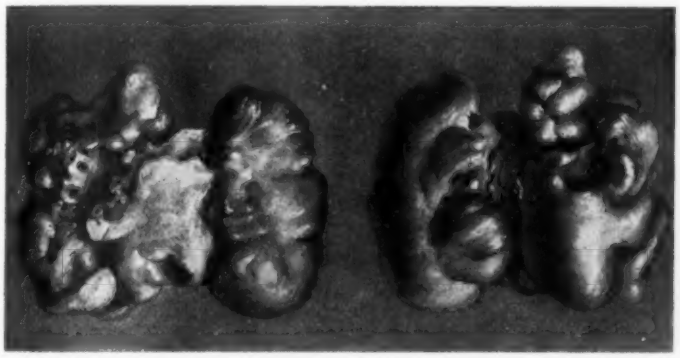

Fig. 5. Drawings of tumor removed.

placed, being attached to the posterior ethmoid, that it was impossible to chisel it loose.

The ethmoid plate was then separated and removed with the tumor, which was done by chiselling all around the dense bone attachment. The mass was finally rocked free, holding it with with overhanging bony lips, thru which the glairy mucus came from the ethmoid cells. The specimen weighed when dry 16.173 grammes and measured 35 x 30 x 17.5 mm. A narrow strip of vioform gauze was placed in the depths of the wound and the skin closed with silk sutures. Rolls of plain gauze were placed

Fig. 6. Result of operation seen from front.

over the upper and lower lids and a firm pad applied over the eye. This dressing was held in position by a flannel bandage. Two days later the packing was removed, skin union was primary and the eyeball freely movable in all directions. The temperature never reached 99.5 and there was neither local nor general reaction.

November 8th, thirteen days after operation, the eyeball was in the same position as its fellow. The vision was 20/15 and disc clear.

Smears made from the mucous discharge showed many polymorphonuclear cells without organisms. The cultures made at the same time showed no growth after four days.

We have been able to observe the boy's face by means of old photographs for several years, and even as late as 1920 there was no gross marked defect. Careful inspection of a picture made in 1921 showed that the left eye was more prominent than the other and divergent. In the consideration of his history we note that he has been mute since birth, altho no other member of the family has been so afflicted. He has had whooping cough, chickenpox, typhoid fever and measles. Wassermann reaction negative.

His ears show marked retraction of clean, thin membranes. He has no air conduction but the low and middle tones are heard thru bone and his voice is typical of one who has never had hearing. The nose shows a deviated septum to the right with large middle turbinates. At no time while under observation has he had a purulent nasal secretion.

Fig. 7. Result of operation lateral view.

It seems striking that altho the osteoma was found to occupy a very large portion of the orbit, the patient was never conscious of annoyance until acute inflammatory symptoms drew his attention to the condition. Altho the left eye was decidedly prominent, on a lower level and divergent, he never complained of diplopia. He was never injured and as far as he knows never had an acute sinus involvement. The most careful and prolonged questioning fails to disclose any responsible factor for his disease.

The examination of photographs made before operation give some impression of the acute inflammatory reaction as evidenced by the markedly swollen eyelids and also the divergence, which altho the eyelid is left in position can be easily seen.

The result justified operation, first because of the marked change in the appearance, second because of the known nature of the growth to increase in size, and third because there was sufficient inflammatory reaction to make it certain that it was only a question of time before he would have another acute orbital infection.

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LIQUEFACTION AND ABSORPTION OF THE CRYSTALLINE LENS WITH ITS POSSIBLE RELATION TO LENTICULAR OPACITIES.

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A summary of the embryology, growth and structure, manner of nourishment and chemistry of the crystalline lens is here given. Views of the absorbtion of lens substance, autolysis in general and liquefaction of the lens substance follow. A proteolytic ferment in the lens, in the aqueous, or both, are the hypotheses discussed. Theories of cataract formation are reviewed and the autolysis of traumatic cataract. Finally treatment to influence liquefaction is discussed. Accepted Membership Thesis for the American Ophthalmological Society.

As the question of liquefaction and absorption of lens substance is so closely associated with its development, anatomy, physiology and pathology, a brief review of some of the salient points is deemed advisable.

EMBRYOLOGY.

It does not seem to be necessary to go into detail of the development of the crystalline lens in that all are familiar with its ectodermal origin from the lateral walls of the anterior vesicle, with later formation of lens vesicle, and the laying down of lens fibers, which finally are compressed centrally forming the central portion or nucleus; while other epithelial cells are formed and cast off, here the cells are in an enclosed membrane, the capsule, therefore, instead of being cast off, they are added to the original tissue.

The development of the capsule continues to be under considerable discussion between those adhering to its mesodermic origin, and those maintaining its ectodermal derivation.

Schirmer and his adherents, as the result of his chemical and histologic studies, but especially the former, believe in its ectodermal origin, as a result of products or secretions of the epithelium of the capsule; the chief reasons being that the capsule is structureless and is easily digested by trypsin, acids and alkalies, whereas connective tissue remains unchanged.

GROWTH AND STRUCTURE OF LENS AND CAPSULE.

Becker estimates the weight of an adult lens as 0.218 gm. and the new born as 0.10 gm., which figures have been confirmed by Priestley Smith.

Clapp in a study of infants' lenses found the average weight slightly less than 0.0953 gm. while adult cataractous lenses averaged 0.1640 gm. Some of these were intracapsular altho the larger number were following capsulotomy. One would expect the percentage of solids to be greater in adults and especially in cases of lenticular opacities; but Clapp's figures show 27.5% solids in infants and 27.02% in adults, which are practically identical and within the limits of error.

Deutschmann and others maintain that the capsule and lens fibers are separated from each other by a layer of albuminous fluid.

The lens capsule is thickest anteriorly (0.016 mm.) being thinner at the equator (0.005 mm.) and posteriorly (0.008 mm.). While some have demonstrated a tendency to lamellar formation in the capsule, there seems to be no evidence of any definite defects or openings in the same.

MANNER OF NOURISHMENT.

The lens is placed in its capsule, which is impermeable to formed elements; and is only indirectly in connection with other portions of the eye, by means of the zonular fibers, which support neither blood vessels nor lymphatics, therefore the nourishment must be derived from surrounding media.

The method and course of this nutritive flow were investigated by Deutschmann, by giving a rabbit one gram potassium iodid in solution and in three hours killing the rabbit. The lens and capsule were placed in a palladium chlorid solution, when it was found that the iodid had impregnated most intensely along the posterior capsule, the equator, and to a lesser degree the anterior capsule, but showed no sign of entering the nucleus or anterior cortical portion.

Ulrich, by injecting ferrocyanid of potassium and subsequently placing the eyes in solution of chlorid of iron and alcohol, seemed to prove that entrance took place at the equator, the posterior capsule remaining unstained. The same results were obtained by using fluorescein instead of potassium ferrocyanid.

Samelsohn, in studying iron particles in the lens, believes the currents to pass from the equator, centrally, then anteriorly dispersing centrifugally towards the zonular fibers, where there are special pores in the capsule for the escape of fluid. Morano claims to have seen these pores, but this observation has not been confirmed.

Bence Jones gave animals, and later cataract patients, carbonat of lithium and showed the presence of the lithium in every part of the lens, this work being confirmed by Bowman and Critchett.

CHEMISTRY OF THE LENS.

The chemistry of liquefaction must, of necessity, be closely associated with the normal chemistry of the lens, which, acording to Neumeister is composed of:

TABLE I.

	Per cent
Water	63.50
Solids	36.50
Albumin	35.00
Insoluble Albuminoids	17.00
Crystalline (b)	11.00
Crystalline (a)	6.80
Albumin	0.20
Fats	0.29
Lecithin	0.23
Cholesterin	0.22
Salts	0.80

The albumins may be divided into two groups, those which are soluble in dilute saline solution and those which are insoluble. These insoluble substances are spoken of as albuminoids, altho manifestly true albumins, as they are entirely dissolved by gastric juice and give all the common color reactions of albumins having the same elementary composition. They are soluble in dilute mineral acids and alkalies, and reprecipitate on neutralization. The amount of albuminoids increase from without inward, with the increasing age of the fibers. Besides the very small amount of serum albumin, the soluble proteids are made up of two vitellins, which are called (alpha) crystalline and (beta) crystalline.

CHEMISTRY OF AQUEOUS.

The compositon of the aqueous is intimately connected with the question

of liquefaction and is given by Halliburton as follows:

TABLE II.

Water	986.87
Solids	13.13
Albumin	1.12
Extractives	4.21
Inorganic Salts	7.70
Sodium Chlorid	6.89

According to Jesner it contains neither fibrin nor fibrinogen. But Claude Bernard, Leber, and others have found traces of sugar, 0.045 to 0.05, which are increased in diabetes. Leber has also shown the presence of a diastatic ferment, which digests starch, with traces of paralactic acid and urea. Aqueous, which is secreted rapidly, as follows repeated punctures of the anterior chamber, differs from the normal aqueous in undergoing spontaneous coagulation and containing a large increase of proteid.

So far as I have been able to find, no suggestion had been made of a proteolytic ferment until 1911, when the work upon "Autolysis of the Lenses" did seem to prove some such ferment.

EARLIER VIEWS ON ABSORPTION OF LENS SUBSTANCE.

Marcus Gunn wrote in 1899 that the lens globulin was readily soluble in sodium chlorid solution, and therefore was dissolved by the aqueous and thus carried off.

Schlosser in 1887 and Schirmer in 1889 studied microscopically the changes taking place when a lens is undergoing liquefaction, but without adding much as to the method.

Parsons in his "Pathology of the Eye" attributes much of the absorption to the action of leucocytes acting as phagocytes and thus bodily carrying away the solid particles.

Kostenitisch also observed lymphoid cells in lens substance undergoing absorption.

Wagenmann has described the presence of giant cells and believes they are active agents in the absorption of lens substance, both by actually ingesting the same and also by furnishing a catalytic ferment, which dissolves and absorbs the same. He also holds that these giant cells have the power to dissolve the capsule itself. Truly a very powerful and convenient cell.

AUTOLYSIS IN GENERAL.

This chemical process was first recognized by Salkowski in 1890, at which time he called it autodigestion. Jacobi in 1900 designated it as autolysis, by which it has been known since that date. It is a chemical process of selfdigestion caused by proteolytic ferments.

The end products of this action are amido-acids, with albumoses, peptones and peptids as intermediate products. But the nature of the ferments is not accurately known. The consensus of opinion seems to be that weak acids accelerate their action, while alkalies inhibit them. Bradley and Morse assume that the action is due to an increase in the hydrogen ion concentration.

The general method of preparation is to free the tissue from blood by repeated washings and grindings, a suspension being made and enough chloroform and toluene added to prevent bacterial changes, placing the suspension in an incubator at 37 degrees C, and making repeated tests as to the amount of the increase in soluble nitrogen or end products.

This process of autolysis has been quite extensively studied in most of the glandular tissues, especially the liver, pancreas, spleen, as well as muscle, brain and other tissues. The first experiments using lens substance were made by Clapp, as published in 1911, and followed by Goldsmith in 1914.

AUTOLYSIS OR LIQUEFACTION OF LENS SUBSTANCE.

In September, 1909, Clapp began the study of the cause of breaking up of traumatic cataracts, with the absorption of the opaque material, as the explanations given in the standard text books did not seem to meet all of the requirements.

Marcus Gunn's statement that the saline solution of the aqueous dissolved the globulin in the lens, certainly was untenable if our chemistry given by Neumeister was correct; and Parsons' explanation that absorption was largely due to leucocytes is un-

satisfactory as microscopic section failed to show a sufficient increase in the phagocytes to justify such a supposition. Fuchs' statement that the fibers swell up and become opaque thru absorption of water, some breaking off into the anterior chamber and being absorbed, altho stating facts, is not especially clear as to mechanism.

One can, with sufficient ease, explain the carrying away of the water and the soluble salts as well as the soluble proteids, but the insoluble proteids, the socalled insoluble albuminoids which make up 50% of the albuminous portion, is the part that requires very decided mechanical means or a change in its chemistry. The mechanical explanation, as previously stated, does not seem to meet all the requirements so, therefore, we turned to the chemical side as offering the most opportunity for solution of the problem.

As soon as one macerates finely a transparent lens in either normal saline or distilled water, there is an almost immediate milky precipitate, which becomes more pronounced on standing, this being made up of broken lens fibers.

After examination of this material it becomes very apparent that this insoluble colloid material must be changed over to some soluble compound before there is much chance for its absorption. The alteration of this insoluble albuminoid to a soluble compound is then the problem before us.

THREE HYPOTHESES DEEMED MOST PROBABLE.

(First) There occurs, in the lens substance or lens lymph, a certain small amount of proteolytic ferment which is capable, after rupture or breaking of the delicate membrane surrounding each of the lens fibers, of digesting this albuminoid to a soluble compound.

(Secondly) The aqueous may furnish a proteolytic ferment which causes solution of these albuminoids.

(Thirdly) A combination of the aqueous ferment and the lens ferment may be the active agent.

In order to clarify the first hypothesis the following experiments were carried out.

Since it has been proven recently that the crystalline lens is organ specific rather than species specific, calves eyes were used as working material; being removed at the abattoir immediately after killing and kept on ice until used, which was always within a few hours. The lenses were then removed, observing aseptic precautions, and each lens macerated with normal saline solution, and the whole made up to 500 cc., and floated between chloroform and toluol to prevent bacterial changes, lightly stoppered to prevent evaporation and placed in an incubator at 36° C. Twenty cc. were removed from time to time and the precipitable proteid removed by Abele's method and soluble proteid determined by Kjeldahl on the filtrate, with the following results, Nov. 18 being the time the experiment was set.

TABLE III.

Variation in Soluble Nitrogen by Abele's method.

Date	N/10 H_2SO_4, cc. required by Soluble Nitrogen in 100 cc.
11/18/09	1.8375
11/22/09	2.5875
11/29/09	2.4625
12/ 6/09	2.5875
12/14/09	2.3275
1/ 3/10	2.4625
2/14/10	3.4625
3/17/10	4.4625
* 5/23/10	7.3675
10/20/10	7.3675

*Set at room temperature until October 20th.

Double estimates were made to assure greater accuracy, and cultures on agar made to exclude bacterial changes.

If there was no fault in the technic, this experiment proved rather conclusively that a chemical change takes place outside of the body, and one can easily believe a similar one could take place within the eye.

The question of proteolytic ferment in the aqueous was attempted by using egg albumin in place of crystalline lens substance, but owing to accident the experiments were spoiled; but M. Goldsmith, by a different method described later, seems to show the presence of such ferment.

The third postulation as to a combination of aqueous and lens was studied in the following manner:

The aqueous was removed from thirty calves' eyes, a one per cent suspension of crystalline lenses made and three tubes set.

To number (one) 5 cc. of lens suspension and 5 cc. normal salt.

To number (two) 5 cc. of lens suspension and 5 cc. boiled aqueous.

To number (three) 5 cc. of lens suspension and 5 cc. unboiled aqueous.

Chloroform and toluol were added to prevent bacterial changes, and the tubes were placed in an incubator at 36° C. for twenty-two days; at which time they were removed and the proteids precipitated by heat and acetic acid, and the nitrogen determined upon the filtrate by the Kjeldahl method, with the following results:

Tube No. 1. Lens and saline 0.8175 cc. N/10 H_2SO_4.

Tube No. 2. Lens and boiled aqueous 1.9675 cc. N/10 H_2SO_4.

Tube No. 3. Lens and unboiled aqueous 2.1674 cc. N/10 H_2SO_4.

The question of accuracy in this experiment is more open to doubt as the quantities used were small and the experiments were not done in duplicate, but if one can draw any inference from the figures, they seem to show very definite action in Tube No. 3, with the unboiled aqueous, while that which had been boiled in Tube No. 2 showed much less digestion, altho even this tube showed over twice as much soluble nitrogen as Tube No. 1, which contained lens suspension only. The explanation of this is somewhat difficult as there should have been a trace more owing to the small amount of albumin and extractives, but the relative large amount requires further study.

M. Goldschmidt, working in Leipsig and apparently unfamiliar with Clapp's previously published work, has arrived at similar conclusions by somewhat different methods.

His first experiment consisted in macerating ox lenses and placing them in dialyzing tubes with frequent washings of distilled water, thus removing the digested portion and determining the amount of soluble nitrogen passed thru. By this method he was able to show at first a very rapid passing into solution, which later became much lessened.

By the Ninhydrin test he seemed to show that there was very little ferment in the aqueous normally, but after repeated puncture this was greatly increased. He later passed threads of elastin thru the anterior chamber of rabbits, and after six hours there was evidence of digestion taking place.

His conclusions were: That the process outside of the body was physiochemical, the aqueous containing a small amount of proteolytic ferment, which was increased by frequent puncture of the anterior chamber. The action of the aqueous combined with autolysis accounts for the breaking up of the lens thereby making it soluble. The rapidity of action depends to a large extent upon the reaction, as alkaline reaction retards, while a slightly acid reaction greatly accelerates the process.

Some have thought that a ferment might be developed in the blood as soon as lens proteid was absorbed, which, in turn, caused further disintegration of lens proteid. Von Hippel, Frenkel, and Nicholas found no such ferment in the blood serum of cataract patients, while on the other hand, Sunseri found a ferment specific for lens proteid in four out of five cases of traumatic cataract.

It seems to us, however, that this ferment in the serum may be the result of the process rather than the cause of the same.

THEORIES AS TO CATARACT FORMATION.

First: *Causes to be found in the Lens itself.* The chief theory based upon this hypothesis is the one advanced by Becke and Deutschmann, which maintains for an unequal sclerosis of the older lens fibers thus putting an unequal strain upon the cortical fibers, with possible impediment in the flow of nutrient fluid. These postulates may be pretty in theory, but as to giving any true explanation they fail to make clear the process in any particular.

Second: *The Chemico-physical theory as advanced by Peters.* This essentially means a shrinking of the lens nucleus, probably brought about by changes in the normal supply of lens lymph. This may be brought about by a spasm or cramp in the ciliary muscle, altering the flow of this lens lymph.

Third: *Condition of General Dys-*

crasia causing Cataract. v. Michel's theory of sclerosis of the carotids is without confirmation, as well as Deutschmann's association with nephritis.

The influence of sunlight, as it occurs in India, has been given as a factor, but the reason for its much greater prevalence in the plains of Northwest India, over that of other parts of India where the sunlight and living conditions are similar, is hardly explainable.

Vossius has called attention to the occurrence of cataract with myxedema, but this as an etiologic factor has not been verified.

Heredity is frequently a decided factor especially in the congenital varieties. Hereditary acquired varieties are most frequently transmitted thru the mother.

Fourth: *Light Waves.* Vogt has succeeded in producing opacities in rabbits' lenses by subjecting their eyes to artificial ultrared rays, and from these findings believes that glass blowers' cataracts are a result of these rays.

Van der Hoeve believes that light, rich in ultraviolet rays causes lenticular opacities by interfering with the nutrition of the lens thru damage of the ciliary processes or their epithelial covering.

DISCUSSION AS TO AUTOLYSIS AND TRAUMATIC CATARACT.

It seems to us that the experiments of Goldschmidt and Clapp show, first, that the aqueous has a proteolytic ferment, which in itself is capable of breaking up albuminoids into simpler compounds, and further, that there is a definite autolytic action of lens proteid itself. Now the possible explanation of the method of causing the insoluble lens proteid to undergo solution with final absorption is somewhat as follows: As soon as the capsule of the lens is ruptured, which is nearly always associated with rupture of some of the lens fibers, there is an entrance of aqueous as well as the lens lymph into contact with contents of the fiber, which had been previously in a sealed package. This immediately causes a diffusion of the soluble proteids with a precipitation and opacification of the insoluble, which, under the combined influence of the autolytic ferment and the proteolytic ferment in the aqueous, undergo a rather slow digestion with production of simpler compounds with smaller molecules, which can pass thru an animal membrane, thus being able to enter the canal of Schlemm or other channels and be carried into the circulation. This action is almost surely a combination of the proteolytic ferment of the aqueous and the autolytic of the lens, nevertheless from our previous experiments, I believe we are justified in the conclusion that this is the mechanism of solution.

MORGAGNIAN CATARACT.

This seems to be explained upon the theory of autolysis much more readily than upon any other hypothesis.

In this condition the capsule is not ruptured, but following the occurrence of the opacity there is a rupturing of the fiber membrane and the beginning of selfdigestion, which proceeds very slowly until finally the greater part of the lens is softened and rendered more or less liquid, while the nucleus being older and more sclerosed remains, settling down in the lower part of the capsule. Altho a large part of the fluid contents are slowly absorbed, there often remains a very dense secondary or membranous cataract. In case the condition occurs early in life, as is sometimes seen, there is no nucleus, but a more or less caseous mass, which cuts like cheese and which finally absorbs to a membranous curtain. While there are a few reports in the literature of a traumatic cataract without rupture of capsule going on to complete absorption, the weak point in these cases is that it would be almost impossible to detect a small rupture far in the periphery, or even posteriorly, by any of our known methods.

THE QUESTION OF SENILE CATARACT.

Many have been the explanations for this condition and possibly the question will always be in a more or less speculative state. The factors which impress themselves most profoundly upon the writer are: That the lens is more or less a tissue that has been extruded or thrown off by the body; that each cell or fiber is surrounded by an animal membrane that is under a certain amount of osmotic pressure; that with variations in the concentration and chemical composition of the surrounding fluid, or lens lymph, this pressure may be so great that a small

rupture in this membrane would occur allowing the nourishing fluid to enter into the fiber with the resulting pre cipitation of the insoluble albuminoids, which seem to be in greater abundance in the nucleus rather than in the cortex of the organ. The condition once beginning would start a vicious circle by swelling, pressure, and change in the concentration or osmotic pressure of the surrounding fluid, thus extending the process.

Just why the lower nasal quadrant is so frequently the first to show beginning opacity will probably be found in the current of the nutrient fluid or in changes in the epithelium beneath the capsule.

Certainly the cases of spontaneous cure, with absorption of the lens, can be explained better by means of this theory than any other.

The question of other forms of cataract might find some means of explanation from these theories, but their number is such that to discuss each variety here would require too much space.

TREATMENT.

Following our experiments as to the cause of liquefaction, there immediately arises the question, that if this is a case of autolysis, does it add any new knowledge that can be used in the treatment of these conditions?

As to the senile and other cataracts of similar nature, where the amount of insoluble albuminoid is large and in a more or less sclerosed condition, it seems that this theory may offer some means of explanation as to their cause, but hardly any for their solution; nor can we see where any of the socalled modern methods of treating incipient cataracts by aiming to arrest their development, will be explained by this hypothesis. The effect of radium in causing lenticular opacities to become stationary, if it occurs, is not explainable on this basis. The question of intermittent use of dionin, glycerin, mercury cyanid, saline, etc., might, by increasing the flow of lens lymph, tend to cause the condition to remain stationary.

In traumatic cataracts, or following extraction in which there is a large amount of soft substance, there does exist a possibility that one may hasten the process. Since it has been repeatedly proven that autolysis proceeds more rapidly in acid solution, some cases of traumatic cataract were given a weak acid solution to use in the eyes at frequent intervals, and in some it did seem as if the absorption of the soft lens was more rapid, but this is difficult to prove satisfactorily. Another point which may be of considerable value is that the proteolytic ferment in the aqueous seems to be in much larger amounts, following frequent puncture of the anterior chamber, so that in view of this fact it would seem that frequent needling with emptying of the anterior chamber would be of value in hastening the process.

The question of introducing a small quantity of an active proteolytic ferment into the lens capsule by means of a syringe or other hollow needle is among the possibilities but hardly within the probabilities.

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NOTES, CASES, INSTRUMENTS

ASTEROID HYALITIS (BENSON'S DISEASE).

JAMES MOORES BALL, M.D.

ST. LOUIS, MO.

In 1894, A. H. Benson, Junior Surgeon to St. Mark's Ophthalmic Hospital, Dublin, under the title, "A Case of Monocular Asteroid Hyalitis", described "peculiar changes in the vitreous humor", the like of which he was unable to find recorded in ophthalmic literature.

Benson's patient was a man, aged 62 years, who had become syphilized in his 32nd year, but otherwise had been in good health and had showed no manifestation of syphilis at the time of examination. With the proper correction, vision for far and near was normal. Benson's description of the ophthalmoscopic findings is as follows:

"The vitreous of the right eye presented a most peculiar appearance. It was perfectly transparent, but scattered all thru it, from close to the back of the lens to the disc, and from the ciliary body on one side to the ciliary body on the other, the whole vitreous was studded everywhere with small, smooth, fixed spheres of a light cream color" . . . "Hundreds of these minute spheres were visible wherever you looked, and they varied in size as much as the stars on a frosty night appear to vary in the clear sky." "It was their remarkable resemblance to the stars on a clear night that suggested to me to call the condition 'asteroid hyalitis'" (*Transactions of the Ophthalmological Society of the United Kingdom, vol. XIV*, 1894, *page* 101).

In the discussion of Benson's paper, William Lang told of a female patient who "had numerous spheroidal bodies in the vitreous, but they occurred in a membrane with many folds, and were not scattered thruout the vitreous. Vision was not much affected".

The President, D. Argyll Robertson, "said he had listened to the paper with great interest because he had had a case exactly similar to that described, with this single exception; that the spots, which were extremely numerous, were loose instead of being fixed, and resembled a series of snowballs". Robertson said he did not know what was the nature of these bodies, but believed that, "they were not cholesterin, for they were not glistening or angular or crystalline, tho of light color; when seen with reflected light they appeared dark."

Since the year 1894, when Benson first called attention to the changes in the vitreous humor which are described above, several writers have reported cases, some of which are similar to, and some are quite different from, the one first recorded.

As regards nomenclature, the writer would suggest that the condition (or disease) be given Benson's name, at least until such time as its true nature may be elucidated.

CASE.

The writer's case was that of a man, J. W. of Wentzville, Mo., a laborer, aged 62 years, whose vision had failed perceptibly for three weeks prior to the date of examination (December 14, 1921). There was no history of trauma; no serious sickness, except two attacks of influenza, occurring three and two years ago, respectively; glasses for reading had been used for many years, and had been obtained from itinerant opticians.

At the time of the first examination, (December 14, 1921), vision was: R. E. =20/200; L. E. =12/200. The pupillary reflexes were normal. The keratometer showed astigmatism "against the rule":— 1. D. in R. E.; and 1.75 D., in L. E. With lenses, vision in each eye = 20/100. Ophthalmoscopic examination showed: R E. = normal fundus, with a slightly pale nervehead; L. E. = fundus normal, with a vitreous humor filled with snowflakes, presenting a picture never to be forgotten.

Like some of the fireworks used in celebrating July the fourth, the snow-white bodies, some rounded and others with a rounded center and with elongated arms, appeared suspended in the vitreous humor, floating up and down or from right to left, in harmony with the movements of the globe. It was noticed that

the remaining teeth—about three-fourths in number—were diseased, and their immediate extraction was advised. Mixed treatment (biniodid of mercury and potassium iodid) was ordered.

Mr. J. W. returned on Jan. 10, '22, with his condition unchanged. It was learned on this visit that he has chewed and smoked tobacco since he was 7 years old.

Several questions arise:

1. Are the "spheres" of congenital, or of acquired origin?

2. Is it not true that, under the term "asteroid hyalitis" two different pathologic conditions have been included?

3. Are not some of the few (14 or 15) cases reported as "asteroid hyalitis", really examples of *synchisis corporis vitrei?*

4. What is the nature of the "spheres"?

At present it seems to be impossible to answer all of these questions. However, from a careful study of the literature it appears that the following conclusions are justified:

1. and 2. That "asteroid hyalitis" includes two entirely different entities: one of which is congenital, and the other is an acquired condition:

3. It is likely that, in several instances, cases of synchysis of the vitreous body may have been misclassed as "asteroid hyalitis":

4. The nature of the "spheres" is known only in the one case (Verhoeff's) which has been the subject of microchemic examination. His patient was a man, aged 74 years, whose right eye, on April 9, 1918, presented hemorrhagic retinitis and asteroid hyalitis. Vision was R. E.— fingers at three feet; L. E. with +2.25 sphere = 20/20. On December 23, 1919, the right eye developed acute glaucoma. Eight days later it was enucleated. Verhoeff found the opacities to "consist chiefly of calcium soaps, calcium carbonat, and certain amounts of fatty or lipoid substances, in combinations of various grades of stability". He believes that, "Their formation is usually if not always dependent upon intraocular angiosclerosis in association with an altered condition of the blood." (*American Journal of Ophthalmology, March,* 1921.)

PROLONGED RETENTION OF FOREIGN BODIES IN THE EYEBALL.

FRANK A. MORRISON
INDIANAPOLIS, IND.

The two following cases are reported as examples of the possibility of long retention of foreign bodies in the eye with preservation of useful vision.

CASE 1. On May 7, 1922, H. G. came under my care giving the following history. When four years of age he was struck in the left eye by a piece of steel caused by one hatchet being struck against another. This was twenty four years ago, he being now twenty-eight. Naturally he would have little or no recollection of events at the time of the accident, or for some time later, and gives the history as given by his parents to him.

He was immediately taken to see a prominent oculist in one of the largest cities of Ohio. His parents were informed a piece of steel had lodged in the eye and a giant magnet would be required to take it out. Treatment was prescribed and four or five subsequent visits made. Each time they were informed of the presence of steel and of its dangers, but no attempt made to extract it. No reason was given. The eye eventually healed well and vision became and remained 20/20ths as shown by records of examination when he entered the railroad service. During these years, however, he had a number of attacks of "inflammation" of the eye said to have been due to "cold" by his local oculist. Recovery was complete so far as vision was concerned. About a week previous to his visit to me in May, 1922, he had one of his usual attacks which seemed more severe and he became alarmed.

An examination upon the above date showed the following: There was a rather mild iritis with but little ciliary involvement if one could judge by the lack of ciliary tenderness. In the superior nasal quadrant, about three millimeters from the corneal margin, was a well defined, slightly elevated black dot about the size of a number eight bird shot, which was taken to be the result of a ciliary hernia.

The tip of a giant magnet was brought slowly to this point and considerable but

not excessive pain provoked. Upon the patient suddenly turning his eye a bright piece of metal shot out from behind the iris, thru the pupil into the anterior chamber. From this position it was easily removed under cocain by corneal incision. The metal was clean and bright, showing practically no evidence of rust, about one millimeter in length and resembled very closely the point of an ordinary sewing needle. Recovery was uneventful, except for the formation of a cataract which is now partially absorbed.

Two interesting features will be readily noted. First the lack of oxidation of the metal in spite of its long sojourn in the eye, and second its position in or at least very close to the ciliary body.

Case 2. J. A. W., fifty-six years of age. At the age of twenty-four (thirty two years ago) was hit in the eye (right) by a small piece of steel. He was taken to a hospital and agreed to accept all risks which were explained to him.

For several weeks his eye was quite painful but eventually recovered, altho vision was reduced to light perception. At intervals he would have attacks of "inflammation" of the eye, but treated them largely himself. Several years after the accident he noted improvement in vision, and was fitted with a glass by an optician. This he was wearing at the time of examination and with it could count fingers at six feet.

At the time of consultation, Sept. 1, 1923, the eyes was intensely painful. The lens had become completely absorbed, while the iris was adherent to the remnants of capsule and old inflammatory membranes. The point of entrance of the foreign body was plainly indicated by a ragged rent in the iris in its upper nasal quadrant.

He insisted an attempt be made to extract the foreign body, before deciding upon enucleation. The tip of the giant magnet was closely applied to the cornea and at once a brownish, scale like mass, seemed to burst thru the iris at the inferior temporal quadrant and spin around in the anterior chamber. Great difficulty was experienced in attempting to remove it thru the corneal incision. It would follow the magnet point nicely but was always arrested at the incision altho the latter was ample. This might be explained by the condition of the particle as shown by the examination after its extraction. The whole mass, 1x½ mm., crumbled easily, except a small bright portion near the middle. It would seem as if the magnetic force had been concentrated upon this small portion causing the mass to rotate and come "broadside" instead of "edge" on. On the other hand the suddenness with which it "burst" thru the iris would seem to indicate it was attracted rather powerfully. However, it is possible it may have, by reason of its long contact with the iris, produced a local atrophic spot in the latter. The recovery was uneventful and vision with the glass remained at fingers at six feet.

CATERPILLAR CONJUNCTIVITIS.

Benjamin H. Grove, M.D.

Buffalo, N. Y.

I saw the patient, a boy of eight years of age the first time, November 3rd, 1923. Six days before, the patient's sister playfully snapped the caterpillar with her finger and it lodged between the lids of the patient's left eye. When I first saw the patient there was a severe purulent conjunctivitis, with edema of the orbital conjunctiva and a very slight haziness of the corneal epithelium. No pain and the pupil reacted normally. The vision of left eye was 20/50.

The eye was washed out with warm solution of boric acid and a weak solution of hydrargyrum corrosivum and permanganat of potash. I prescribed argyrol 20% for home use. The next day, November 4th, there was a decided abrasion of the corneal epithelium about 3 mm. in diameter occupying the center of the cornea, and the whole upper half of the corneal epithelium was mottled and macerated. November 6th eye some better. November 7th no more purulent secretion and but little edema of the conjunctiva. All the surface of cornea was normal, except the central abrasion, which was contracting. Daily the condition improved, and on November 14th fluorescein showed a smooth and normal corneal surface. The vision of the left eye was less than 20/20. I venture to report this case because it is the first in my experience and I imagine it is somewhat rare.

SOCIETY PROCEEDINGS

Reports for this department should be sent at the earliest date practicable to Dr. Harry S. Gradle, 22 E. Washington St., Chicago, Illinois. These reports should present briefly scientific papers and discussions, include date of the meeting and should be signed by the Reporter or Secretary. Complete papers should not be included in such reports; but should be promptly sent to the Editor, as read before the Society.

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

Section on Ophthalmology.

October 11, 1923.

DR. MCCLUNEY RADCLIFFE, Chairman.

Practical Aspects of Irregular Astigmatism.

DR. EDWARD JACKSON of Denver, in opening his paper, stated that he found it a source of great satisfaction that he could still count himself a nonresident Fellow of the College; and he could not find a more pleasant or profitable place to come when within reach of Philadelphia. He said that he had tried to live up to his opportunities by occupying for several days one of the study rooms in the College building. With reference to some subjects which seem to be in the outlying territory of refraction, perhaps the best that can be done with them is to show that they are of practical importance so that they thereby command attention. He read a paper which later will be published in full in this JOURNAL.

Discussion: DR. EDWARD A. SHUMWAY said that as usual Dr. Jackson had given in his paper something new and worthy of close attention, and it was difficult in the short time allotted to add much in the discussion of the paper. The two points to which he particularly calls attention are symmetric aberration and irregular astigmatism in general. As he has well said, aberration is a comparatively common finding when the pupil is widely dilated, and may be positive or negative.

Positive aberration is most frequent, and may be often demonstrated by retinoscopy, especially in hyperopic eyes, in which, if a small sharp light is employed, a central shadow moving with the movements of the mirror will be present long after the peripheral shadow has been reversed by the lenses placed in front of the eye. This is a point which frequently upsets the beginner's judgment in estimating the refractive errors by this method.

Dr. Jackson's case of negative aberration with a difference of 16 D. is unusual, altho myopia is the common accompaniment of advancing nuclear cataract. The practical point, is of course, the necessity for a postmydriatic test in all cases, especially when the refraction of the central visual area differs so markedly from that of the periphery. Patients will not be comfortable if they are not ordered the correction to be used when the pupils have returned to their normal size. The same thing holds true in all types of irregular astigmatism, particularly in conical cornea, to which he has drawn attention.

Dr. Jackson's suggestion of the elimination of the diffusion images which are noticeable if the pupil is dilated by the mydriatic, or in a dim light, is a clever one. Passing a card parallel to the second image, especially when two parallel lines are present, will, as he says, cut out one line, which is due usually to the myopic periphery, and will relieve the patient's concern as to the lack of sharpness of the images, even after correction of the regular astigmatism; altho many patients are more confused by a scientific explanation than if they are simply told that the eye whose vision cannot be brought to normal, is a "lazy eye".

His suggestion that, before doing an optical or visual iridectomy, or in the choice of a glass for reading in the presence of opacities of the cornea, a careful search be made not only for clear areas on the cornea but those which may be best corrected by a lens, should be especially emphasized.

History of Study and Practice of Refraction in Philadelphia.

DR. HOWARD F. HANSELL stated that for the past one hundred years Philadelphia has been the home of the professional refractionist. Before the days of specialists the general surgeon made ophthalmology an important part of his work, devoting no little time to the correction of errors of refraction. It is recorded that John McAllister, an op-

tician of Philadelphia, made and fitted a plano-convex cylinder as early as 1828, probably the first attempt to correct astigmatism in the United States, but whether on his own initiative or from a prescription is not stated, probably after many consultations. The men who unconsciously founded the Philadelphia School of Ophthalmology, Dyer, Thomson, Harlan, Norris, McClure, and who established themselves as oculists, placed the study of refraction and the correction of optical defects on a permanent foundation. Under the initiative of these pioneers the correction of refraction was and still is considered of vital importance, and was carried on with greater nicety and care in Philadelphia then in most other cities. Dr. S. Weir Mitchell worked in close collaboration with the oculists, and indeed stimulated them to find in the eye causes of nervousness and other reflex symptoms. Evolution from general surgeon to specialist was slow and beset with many difficulties. Finally ophthalmology was placed on a solid footing and became a recognized specialty. Dr. Hansell concluded the paper with mention of the principal eye dispensaries of the early days and a brief statement of the great value to the profession of the Section on Ophthalmology of the College of Physicians.

Dicussion. Dr. George de Schweinitz, after expressing his appreciation of Dr. Hansell's paper, made a brief reference to Dr. James Wallace, for many years associated with Dr. William F. Norris's clinic in the University Hospital. His intimate knowledge of the refraction of the eye and of physiologic optics, of which a number of the physicians in the audience were beneficiaries, should not be forgotten in recalling the work and worth of Philadelphia ophthalmologists. Dr. Wallace, he said, possessed a truly "mathematical mind," and few men, if any, among those devoting their attention to ophthalmology, were his superiors in this respect. Unhappily, his health declined at a comparatively early period in his career, and he was unable to carry on his important work.

Dr. S. Lewis Ziegler said that the early days of refraction in Philadelphia were mostly traditions, confirmed by observation and personal contact with some of the active spirits of the time. Credit was given Drs. Dyer, McClure, Thomson, Norris, Harlan, Risley, Randall, Jackson and Oliver as having had much to do with the early history and development of what has been known as the "Philadelphia School of Refraction". As Dr. Hansell has stated, many of the older men gave particular credit to Dr. W. W. McClure, an early surgeon of Wills Eye Hospital, as being one of the pioneers chiefly instrumental in bringing about the development of refraction, (1) by making some of the earliest tests for astigmatism, (2) by amplifying the fractional units of cylinders below one diopter, and (3) by constructing models of the eye for the purpose of teaching refraction and eye diseases to members of the medical profession, who crowded the old clinic room of the Wills Hospital to hear his course of lectures. It was stated that the envy of one of his colleagues caused public teaching to be banned for many years by resolution of the Board of Managers. This early rebuff reacted strangely on McClure and made him very reticent. He never published any of his original work, and hence his fame was soon eclipsed by his more active colleagues. He was the first to refract cases of high hyperopic astigmatism, and his success in correcting the high error of Dr. Thomson turned the latter's attention to this type of ophthalmic work. McClure was later tendered the chair of clinical ophthalmology at Jefferson College, but, having acquired a distaste for public teaching, he passed this offer on to Thomson, who was later elected to that position.

McClure's early trial case was weird but workable, being made up of a variety of lenses both in size and style, in order to fill out the sequence with fractional numbers of one-eight of a diopter. At a later date he adopted the trial case manufactured by Ivan Fox. His teaching models of the eye were frequently borrowed by Norris to use in his annual demonstration course at the University of Pennsylvania. It is said that in the early days McClure often laid out his cylinder

lenses, fastened them on a sheet of paper, marked the axes, and carried them down on a tray to Borsch to be ground. One day while discussing these problems with Borsch, it was suggested that a protractor would obviate his trouble, and so he added this to his trial frame, after which it was only necessary to write out the formula of the trial lenses in order to have them duplicated.

Dr. Ziegler said that the trial cases made by Nachet, of Paris, were the earliest he had personal experience with when he began refraction work in 1886. The axes and lens numbers were scratched on the surface of the glass. This case was supplanted about 1887 by Ivan Fox's trial case with the diamond shaped metal handles that had the numbers stamped on them. Dr. Jackson also devised a case of extra small lenses that were manufactured by Queen & Co. Dr. Ziegler's early interest in heterophoria led him to devise a wide metal cell to hold the prisms, as it was impossible to do accurate work with the older types. The first set was made by the American Optical Co., in 1893. The following year he had the Fox Optical Company make up a complete case of trial lenses mounted in the same type of wide metal cells. These comprised eighths of a diopter up to 2 D, quarters to 7 D, halves to 10 D and single diopters up to 35 D in sphericals, or 53 pairs in all. The same sequence in cylinders reached 30 D, or 41 pairs in all. The prisms were in pairs to 6°, and in single numbers to 40°, or 34 in all. A modification of this trial case was later adopted as the Wills Hospital standard type. He also devised a trial frame to hold four of these lenses, with wide metal cells, which he has used with great satisfaction during the past thirty years and without a single item of repair. A prism exercise frame was also constructed, with a locking device to hold the metal cells in position, thus preventing rotation of the axis.

Dr. J. Milton Griscom said he only wished to emphasize two general points. Successful refraction is dependent not only on a thoro knowledge of physiology and optics, but also upon the exercise of good judgment in determining the relative value of each of the factors which made up a complete examination. It is not enough that one should measure with accuracy the static refraction in every case by means of the retinoscope, trial case or other method. With this as a basis, the other factors determined by a complete study of the accommodation and the extraocular muscles, together with the age, occupation, general health and temperament of the patient, should each be given careful consideration before a prescription for glasses is given. It is also of the greatest importance in addition that after the glasses are ground and placed before the patient's eyes, the ophthalmologist shall personally see that the adjustment is accurate. He believed in most instances where bifocal glasses are uncomfortable it is because they are not properly centered with regard to the upper or lower segment.

When one considers that from 75-85% of most ophthalmologist's private practice consists of refraction, it would be only reasonable to assume that those specializing in ophthalmology would give a good proportion of their time and effort to this branch of work. In actual practice, however, for several years it has been increasingly difficult to find a student or young practitioner who was willing to devote at least half of his time in clinical work to the study of refraction. A world wide traveller and trained observer recently wrote, that after a survey of world conditions he was forced to conclude that people in general were trying to find a substitute for work. This may explain the tendency evident among certain entirely ethical and able men to advocate refraction by short-cut methods, and the unwillingness among students in general to spend any more time on refraction than they have to. Be that as it may, the fact remains that there is no royal road to thoro and scientific refraction. Patient, painstaking, persistent work is the element without which nothing is possible.

Dr. Edward Jackson said there were two things that have not been brought out here that are of interest to the Section. Dr. Ezra Dyer was working with Donders and Snellen in 1861. On Donder's suggestion, Snellen took up the problem of test types. Dyer returning to Philadelphia had printed a card of test type in 1862, some months before Snel-

len. Snellen's work was more complete, went into mathematical principles, sifted letters to be used for that purpose; and along with his little brochure on the subject of testing acuteness of vision he put out his test, which became known all over the world. Dyer's type was practically the same as Snellen's type, and he always gave Snellen credit for the principles applied in their construction.

In Philadelphia at that time the feeling of certain older men, who were in the practice of general surgery, against specialism, prevented Dr. Dyer's work from being properly recognized from the time that he returned to Philadelphia for several years thereafter. He was very much disappointed when he could not get elected to Wills Hospital, a coveted opportunity he only attained years later.

The other point is the story of how the first test case was secured at Wills Hospital. Dr. Charles Hermon Thomas was the resident at Wills Hospital. One day a woman about forty years of age came in who had been doing fine needlework for many years, and her eyes had given her a great deal of inflammatory trouble. She had gone to one of the most famous general surgeons in Philadelphia, and he had treated her eyes week after week with bluestone without relief. Dr. Thomas, after hearing her story, went to McAllister's and borrowed a trial case, worked out five diopters of hyperopia and had glasses made for her. With these lenses she was able to continue her work with comfort for many years. With that story Dr. Thomas went to the Hospital Committee, and the first trial case was purchased for Wills Hospital. That was the story from Dr. Thomas.

The Technic of Refraction and the Prescribing of Glasses.

Dr. S. Lewis Ziegler called attention to the unique advantage to be obtained by a study of the practical methods of different refractionists. He always employs a cycloplegic under 45 years, and often up to 50, or even beyond when necessary. Homatropin, hyoscin and atropin are his cycloplegics of choice. Homatropin hydrobromat gr. i to ℥ss is instilled night and morning, and every ten minutes for an hour, preceding the test, or ten times in all. The hyoscin and atropin are instilled t.i.d. For many years he has used cylinder fogging in all tests for astigmatism, in order to locate the axis quickly and accurately. The plus cylinder should be at least 1. D. stronger than the supposed astigmatic error. By rotation of the cylinder the vertical lines will lean over to one side or the other, and the axis will thus be revealed. This supplants the stenopaic slit. The usual refraction with trial lenses is then completed. The test case must be the final arbiter in all cases.

Retinoscopy has proved most useful in children, in illiterates, and in proving up the completed refraction, by adding sph + 1.D. and proving the shadows neutral. Javal's keratometer is valuable in differentiating between corneal and lenticular astigmatism, or in aphakia. He always utilizes the 6 meter range, or a short 5 ft. range if the vision is very poor, changing to the long range as the vision is improved by the lenses. His test card for near vision is based on the Jaeger reading types, to which is added pin points, needle holes and a bar of music. Full cylinders are usually ordered, but a slight reduction may be necessary in the high numbers. Cylindrical distortion and anisometropia must be carefully adjusted and the heterophoria corrected. Mixed astigmatism should be corrected by full cylinders crossed at right angles and not spherocylinders. A slight spherical reduction may be made in prescribing. Irregular astigmatism is a problem by itself to be carefully worked out and ordered as found. Traumatic astigmatism often falls into this class.

Changes in refraction may be frequent and disturbing. If they cause asthenopia the cycloplegic test should be repeated. In any case refraction should be repeated every year or two in myopia, every two or three years in hyperopia and at similar intervals in presbyopia. All tests should be made two days in succession, to confirm the result.

Postcycloplegic tests are made in the majority of cases, and the prescription based on all of the units revealed by the various tests. Full spherical cor-

rections are seldom prescribed. Sometimes a reduction for distance is made but a corresponding increase is ordered for near, thus giving bifocals for young persons. In the high hyperopia of children a partial correction is first ordered and an increase made when the eyes have become accustomed to it.

In hyperopia a reduction of 0.25 D. to 2 D. is made in corrections ranging from sph + 0.25 D. to sph + 8 D. In myopia a full correction is ordered up to sph — 5 D. From that amount up to sph — 20 D. an allowance of 1 D. to 8 D. is made for near work, or more if necessary. Myopic corrections are prescribed in ground bifocals, with the lower field centered and the upper one deviating slightly. The bottom of the curve is located 3 mm. below the geometric center of the lens.

For cataract patients convex torics are ordered, with centers displaced down 3 mm. and the bifocal segments set next to the eye. These are sufficiently prismatic, base down, to bring floor objects up into the field of vision and thus make locomotion more comfortable.

In monocular aphakia the distance spherical is reduced one-half in order to obviate the anisometropia. The regular amount of bifocal is added to both eyes, if needed. If one eye is aphakic and the other still cataractous, the far and near correction can be ordered in a reversible frame. In young aphakics one eye is sometimes corrected for distance and the other for near. When the average case of aphakia is rapidly neutralized by progressive myopia, no glass will be required. Aphakic accommodation is rare but may occur in cases of this character. A correcting lens may then often be omitted.

Heterophoria is measured by the old-fashioned Graefe test, the displacing prism being placed before the right eye and a red glass over the left, with the Greek cross test-object located at a distance of 20 feet. A small black Greek cross, printed on cardboard, is used for the near heterophoric test. The final prism measurement is made over the correcting lenses and may be worn in the prism trial frame before prescribing. The order for prisms may be divided between the two eyes if over 3°. In correcting hyperphoria the prism is preferably ordered base down because more comfortable. In hyperphoria the total error is prescribed, in esophoria two-thirds of the error, and in exophoria one-half of the error. The latter correction can often be omitted. Other exceptions may occur. The glasses should be returned for neutralization on the prism scale and for advice as to fit. At the end of one month the patient should return for examination and adjustment of any psychologic annoyance that a new environment may inflict.

Observation on Refraction.

Dr. P. N. K. Schwenk said, that in examining the records of 21,812 patients he found 18,268 or 83.7% came for refractive errors; 440 or 2% refused drops. These figures illustrate the relative importance of refraction. Good refraction is not common because the students of the day shun it as being irksome and tedious. For the best refraction the patient, duly impressed with the importance of getting the most accurate findings, must be comfortable and at ease while the doctor works with perseverance. Complete ciliary paralysis is essential in obtaining accurate results. Since 1887 he had not used homatropin because of its failure to produce complete paralysis of accommodation, its variable strength, and the uncertainty of securing the proper drug. Since this time duboisin has been used in all cases under 42 years of age.

His procedure is to take the family and personal history and note the general physical condition. The visual acuity is recorded and an external examination of the eye is made. Duboisia (gr. 1/8 to f3ss of dist. water) is then ordered, one drop to be put in each eye the night before and the morning of coming, and the wearing of dark glasses. The drops are to be repeated at noon, in the evening and the next morning, two refractions being done on two successive mornings. To instill the drops the patient is instructed to have the outer 1/3 of the upper lid pulled up and have one drop of duboisia pass under the lid. In this way the drop will pass over the cornea

thus insuring maximum absorption. Duboisia, like atropin, causes muscular depression together with delirium, but there is no danger when used as directed with a pointed dropper. The beaded dropper should be condemned as it gives too large a drop. With cycloplegia complete the shadow test, fundus examination, refraction and muscle tests are made.

All cases of over 42 years of age are refracted without a cycloplegic, a weak mydriatic being used for the fundus examination when necessary. A correction is given to all patients having a simple hypermetropia of 1 D. or more. Below this, correction is given full for near work. Any astigmatic error, be it ever so little, is corrected fully with as much of the sphere as suits the case. Eserin is not instilled after the cycloplegic as a rest for the eye is indicated.

The glasses prescribed should have the lenses fixed in the frame to prevent any rotation of the astigmatic axis. In hyperopes the full cylindrical with a spherical deduction of from 0.50 D. to 1. D., according to the degree of hypermetropia, is ordered. In myopia a full correction is prescribed in all cases. When the error is 5. D. or more, a plus 1. D. bifocal is added, if 8. D. a plus 1.50 D. bifocal and so on. Low myopes are given a little over correction to excite convergence.

Muscles are tested in all cases. Prismatic correction is ordered for an esophoria over 2 degrees, or an exophoria over 4 degrees. Lateral exercises are practiced in exophoria. Hyperphoria is corrected up to 6 degrees and fusion is obtained. After cataracts have been extracted from each eye, there is invariably heterophoria present which can be relieved by prisms. A sudden increase of hypermetropia in adults should be looked upon as a premonitory symptom of brain tumor. Dr. Schwenk said he had three such cases on record. He has noted an increase in hypermetropia in retinal and choroidal disturbances.

As an instance of rare refraction, Dr. Schwenk gave the following:

O. D.—16 cyl. axis 10°=20/50.

O. S.+6⌒—16 cyl. axis 150°=20/50.

He wore this correction for 21 years when the following was given:

O. D.—18 cyl. axis 5°=20/50.

O. S.+6⌒—21 axis 130°=20/50.

Full correction was ordered with plus 3 D. bifocal.

Discussion. Dr. G. Oram Ring said that when the chairman of the Section expressed his desire to have the speaker outline upon the critical canvas of the hour, the subject of present methods for the correction of refractive defects and their associated muscle imbalance, he gave voice to the conviction that we had fallen from our former high estate in the practice of this important division of our special work, which for the average oculist constitutes more than fifty per cent of his consultation hours.

Dr. Ring said that many of the patients who gain entrance to our consultation rooms are of those moderns who feel the driving urge of the insensate craze for speed, that finds its expression in a demand for the very briefest of examinations. The state itself, in this matter at least, an unwise guardian, is licensing a group of men called optometrists, not permitted to use cycloplegics, not trained to scientific ophthalmology but quite sure that cycloplegics are an unnecessary hardship upon the patients, and who are using every possible effort to propagate their credo. Of such now practising in our state, there are eleven hundred and upwards, over 400 being located in and around the city of Philadelphia. The outcome of such methods you all see far too frequently to here need elaboration.

Dr. Ring said he was in accord with Dr. Schwenk regarding the desirability of the longer cycloplegic, and constantly emphasizes its importance, especially to the parents of his young patients. Following the teaching of Dr. Risley, he had for years used hyoscyamin sulphat, and had never noted any significant constitutional disturbance following its use.

In only one instance in a long hospital service did he recall where a cycloplegic induced a glaucoma. His chief of clinic ordered an assisting interne to write for the patient a solution of boric acid, and she was given a solution of atropin, which she used as an eye wash. Such a circumstance does not vitiate the thesis. It was simply an inexcusable error.

It is the routine practice in his office

in using a cycloplegic in refracting a patient at or around forty years of age, to invariably take the tension with the tonometer. He did not limit the cycloplegic for refractive purposes to forty-two years of age. In using homatropin he instilled a drop every fifteen minutes for an hour and a half, and made the refraction about thirty or forty minutes later. This is often supplemented by the instillation of a drop the previous evening and in the early morning.

Fig. 1. Epithelioma of lower lid showing lesion reacting to radium.

Fig. 2. Dr. Bane's case after radium treatment.

As to the frequency of examination in hyperopic cases, there is no hard and fast rule, but he would certainly suggest having the eyes looked after every few years. This is especially important in the lower degrees of hyperopia and hyperopic astigmatism in children during the formative and even more advanced educational period, because of the frequency with which the eye becomes myopic.

A postcycloplegic test is routinely made, and it is of first importance to make a careful muscle study with the new correction in position and base the management of the case, from the viewpoint of muscle imbalance, upon such findings.

In conclusion he suggested: First, that we emphasize more and more the importance of the cycloplegic, if proper refractive results are to be achieved. Second, that we increasingly insist upon the longer cycloplegics rather than the more or less unfortunate and often too casual use of homatropin; third, that a properly prepared and carefully applied solution of homatropin is of value especially in our adult cases; fourth, that, whether or not we have made preliminary muscle studies, such studies should invariably be made as a part of our postcycloplegic tests and our judgment as to the muscle management of the case be based upon such examination.

C. E. G. SHANNON, Clerk.

COLORADO OPHTHALMOLOGICAL SOCIETY.

October 27, 1923.

DR. E. M. MARBOURG, presiding.

Epithelioma of Eyelid.

W. C. BANE, Denver, presented a man, aged sixty-three years, who had come on account of a growth on the left lower eyelid of which he had been aware for two months. The eyelid, however, had been annoying him for about eight months, the symptoms being a burning sensation and occasional discharge. There was no history of preceding injury. Running inward from the margin, at the center of the inner surface of the left lower eyelid, was an elevated corrugated whitish growth, five by six mm. in diameter, and elevated 0.5 mm. above the surrounding area. The tissue between

the growth and the scar surface was quite hard. The growth had been treated with radium on October 26, and the tissues were in the beginning stage of reaction to the radium.

Retinal Tuberculoma.

A. C. MAGRUDER, Colorado Springs, presented a man, aged twenty-five years, who had come complaining of cloudy spots before the left eye, which he connected with getting soap in the eye and subsequent rubbing. He also stated that for ten or twelve years there had been a large spot before the left eye. The vision was, R. 20/30 plus, L. (corrected) 20/70. The pupil of the left eye was about two-thirds dilated, and this eye was distinctly harder than the right. There was no pain. To the nasal side from the disc was a grayish mass which gave the impression of being either detached retina or retinal exudate. The patient had come to Colorado Springs eight months previously, after a very small pulmonary hemorrhage.

Discussion. EDWARD JACKSON, Denver. Tuberculosis seems the likeliest diagnosis at the present time. I did not see any intraocular hemorrhage. As between an extensive area of atrophy and a large whitish mass I am inclined to the latter, altho the vitreous is so hazy that it is impossible to see details which can be accurately measured. It would probably be worth while to use small therapeutic doses of tuberculin, watching cautiously for reaction. With widely dilated pupils and very strong light it might be possible to obtain fairly distinct details of the growth.

W. C. FINNOFF, Denver. My impression was that this was a mass of exudate into the vitreous, in front of the vessels. It seemed to me that there was pathology in the other eye, altho the pupil was small and the fundus not easily visible.

Eye Destroyed by Popping of Cork.

E. R. NEEPER, Colorado Springs, presented a woman, aged fifty-one years whose left eye had been injured in June last, by a cork popping from a large bottle of peroxid of hydrogen. The evidence of internal injury had included much conjunctival edema and the filling of the anterior chamber with blood, and the vision of the injured eye was counting fingers at six feet. The eye had remained painful for many days, and fresh hemorrhage into the anterior chamber had been noted daily for two weeks.

After four weeks the anterior chamber had cleared greatly, and a mass of material of brick-dust color and heavier than the aqueous had appeared in the anterior chamber for a few days. This material seemed to enter the chamber thru the ruptured suspensory ligament at about 120 to 130 degrees. The base of the iris was still slightly tremulous in this region. From above and about the macula, an irregular broken line of pigment patches extended to the upper inner part of the ora serrata, and just back of the ora serrata in this region was visible a whitish patch about three disc diameters across and irregularly square in shape. The vitreous contained hemorrhagic débris. The aqueous and lens were clear, except that there were red tags within the pupillary area.

Discussion. W. C. FINNOFF, Denver. I saw this patient with Dr. Neeper last July. At that time she had good light perception and projection. There was a fine dust like deposit, red in color, on the surface of the anterior lens capsule. Three fine red strands, which resembled bloodvessels, radiated upward in the sector of the pupil between ten and eleven o'clock and disappeared under the pupillary margin of the iris. With magnification, they looked like blood clots. The fundus has cleared up very decidedly. When I saw it last, all details were obscured by the hemorrhage. I believe that the blood in the anterior chamber came from ruptured vessels in the ciliary body and not from the iris.

EDWARD JACKSON, Denver. This case may become more interesting still as the vitreous clears further. I thought first of contrecoup, but Dr. Neeper's description renders it probable that the injury was more direct in character. Years ago I reported such a case in which there was apparently ordinary rupture of the choroid, with a

series of light yellowish spots concentric with the disc. These spots in the choroid were so prominent that they showed thru the clouding in the vitreous. The patient had been struck with a marble or small stone. In a week or so the spots of choroidal exudate disappeared, and the man afterward had a normal fundus. The apparent rupture of the choroid was probably due to exudate, with actual hemorrhage from the choroid escaping into the vitreous.

Senile Neuroretinal Changes.

E. R. NEEPER, Colorado Springs, presented a man, aged eighty years, who had come on account of an optician's diagnosis of cataract. Vision had been failing for years. There was atrophy of each optic disc, with an atrophic ring surrounding the disc. Thruout the fundi were disseminated red, white and pigmented patches.

Retinal Detachment.

E. R. NEEPER, Colorado Springs, presented a woman, aged sixty-four years, who had come originally in September, 1921, on account of two attacks of semiblindness in the right eye during the previous twenty-four hours. At that time the pupil was dilated and the vitreous hazy, probably from a small hemorrhage. In five days the vision of this eye had risen to 20/40, but there was little further change in vision during the next twenty months. A somewhat similar attack in May, 1923, was followed by persistence of heavier débris in the vitreous. After a further attack in September, 1923, the vitreous remained cloudy for some time, and after three weeks a large detachment of the retina was visible and vision was 20/200. The vision of the left eye had been steadily 20/40.

Intraocular Foreign Body.

E. R. NEEPER, Colorado Springs, presented a man, aged thirty years, who had originally come in April, 1909, on account of injury to the left eye by a splinter of metal, which had broken off while he had been hammering a chisel five days previously. At that time there was a corneal scar near the limbus, the central lens was cataractous, and vision was light perception. Altho examination had shown the foreign body in the posterior vitreous, surgical interference had been declined. The patient had reappeared for the first time in October, 1923, on account of a small foreign body on the right cornea. The left eye had an active dendritic ulcer, and the whole cornea stained faintly. There were four points of scar tissue in the cornea as tho from previous attacks of keratitis. The vision of the injured eye was doubtful light perception. Should this eye be enucleated, and what was the danger to the other eye?

Discussion. W. C. BANE, Denver, felt that the safest thing to do was to enucleate the left eye.

EDWARD JACKSON, Denver. The danger of sympathetic inflammation in the right eye is probably not imminent, but sympathetic irritation has begun, sooner or later the left eye will have to come out to relieve it, and there is also some danger to the right eye from later sympathetic inflammation.

W. C. FINNOFF, Denver. The left cornea seems to be steady and the tension increased, and this probably accounts for the corneal changes.

W. H. CRISP, C. E. WALKER, and J. A. MCCAW agreed that the eye should come out.

Posterior Polar Cataract.

E. M. MARBOURG, Colorado Springs, presented a man, aged forty-five years, a printer by occupation, who had come on account of failure of vision of the left eye, apparently due to lead poisoning. The right eye had a posterior polar cataract. This eye had been struck with a stick when the patient was a boy, and its vision had been poor ever since.

WM. H. CRISP, SECRETARY.

OPHTHALMOLOGICAL SECTION OF THE BALTIMORE CITY MEDICAL SOCIETY.

Nov. 22, 1923.

DR. J. W. DOWNEY, Presiding.

Iris Inclusion for Glaucoma.

DR. CLAPP showed two cases. A white man of 60 years of age was op-

erated upon five months ago for acute glaucoma with retinal hemorrhages by iridotasis, with tension remaining normal since operation.

A colored girl with simple chronic glaucoma with tension of 60. Three weeks after the operation tension is reduced to 40, but fields have not expanded.

Indications for Various Operations in Glaucoma.

Dr. Wm. Zentmayer, Philadelphia, said the relatively few cases seen by any one surgeon compel us to base our judgment in part on the statistics and opinions of others. The constant causative factor in secondary glaucoma is obstruction to the filtration angle. This is presumptive evidence that the same cause acts to produce it in primary. The most important etiologic factor is age with its sclerosing tendencies, and increase in the size of the lens.

The choice of operation is influenced by the type and stage of the disease. A basal iridectomy is the only operation to be considered in acute congestive glaucoma. The cause of the attack is probably some acute disturbance of metabolism or vasomotor control. Subsequent systemic disturbances of this nature fail to produce hypertension because of the permanent opening of filtration by the iridectomy. In subacute or chronic congestive glaucoma, effectual iridectomy is difficult to secure. A filtering scar operation, or a cyclodialysis is preferable. If an operation is decided upon in the chronic simple type we have the choice of several operations, all of which have to do with the iris and this probably results in freeing some portion of the angle.

The objections to the Elliot operation are that iritis too frequently occurs; that secondary changes, usually in the development of lenticular opacities, sometimes occur, probably the result of hypertonus; that late infections are relatively frequent.

The operation of iridotasis is too new to judge. When we have the statistics of a large number of operations, we shall probably find that late infection is as frequent as after trephining. There is also the danger of sympathetic ophthalmia. Cyclodialysis is satisfactory as a preliminary operation and sometimes gives permanent relief. The result sought to be secured in a peripheral iridotomy can better be obtained by an iridectomy.

Iridectomy is still the operation of choice, except in the presence of greatly contracted fields, where trephining should be selected, until it can be determined whether the claim for it, that it does not produce a loss of fixation, is based upon fact.

In congenital glaucoma, unless the case is seen very early, posterior sclerotomy is the safest and probably most efficient operation.

In glaucoma due to obstruction of the central vein of the retina, cyclodialysis is the logical operation, because of the usually gradual reduction of tension following its performance.

In cyclitic glaucoma, paracentesis of the anterior chamber, keeping the wound open by the introduction of a spatula, permits the continuance of a cycloplegic.

In occlusion of the pupil, iridectomy is to be performed, unless iris bombé has developed, when transfixing of the iris is indicated.

Discussion. Dr. E. A. Knorr discussed various iris inclusion operations. The late Dr. Harlan had performed several at the Presbyterian Hospital and was favorably impressed with the results. In six cases reported, four showed decreased tension, and three had relief from pain. No improvement of vision occurred in any of the cases.

Dr. Theobald. Just one observation made to me by Mr. Higgins of Guy's Hosp., London, many years ago "That a badly performed iridectomy reduced the tension better than a classical one." This conforms to my experience, and for several years I have not been particular about replacing all of the iris pillars.

Dr. Friedenwald. This is probably the most difficult problem in Ophthalmology. My own experience is that iridectomy is best for acute glaucoma, while in simple chronic glaucoma, I

rely entirely upon the trephine. In a few cases I have been greatly disappointed as the tension has remained high.

DR. CROUCH: My experience with the trephine has been very unsatisfactory, late infection, quiet iritis and hypotension being the complications seen. I still believe that the iridectomy is the operation of choice, even in simple chronic glaucoma if taken early, as this has stood the test of time. As to iridotasis, I have recently performed this operation in a case where one eye had been lost, the patient having a family history of previous glaucoma. The case was acute with very high tension and was followed by reduction of tension to normal and 20/30 vision.

DR. MCCONACHIE: My experience in simple chronic cases is that they all do badly if watched a sufficient length of time.

DR. HIRAM WOODS: I have seen an acute glaucoma develop while examining the eye, with dilation of pupil, marked increased tension and injection. I should like to know how one is to tell when starting to treat a case of acute glaucoma, whether it is fulminating or not. Some cases of glaucoma stand tension, with no decrease in vision or fields, while in others a very slight rise of tension may cause both contraction of fields and diminution of vision. Cases with friable iris are most difficult to operate upon. I believe that an iridectomy, all in all, is the best operation. Trephining has not been satisfactory in my hands.

DR. TARUN: Reduction of tension in glaucoma is not so important as retention of fields and acuteness of vision. The tension may vary greatly. Iridectomy is still the operation performed universally the world over, and in my opinion is superior to all others. I believe a more satisfactory iridectomy can be done with the Graefe knife than with a spade knife.

DR. ALAN WOODS: In reference to the danger of sympathetic trouble developing from incarceration of the iris, I have seen no such cases and am unable to state whether the danger is great or small.

DR. ZENTMAYER in closing said: A badly performed iridectomy probably is efficacious because the iris is removed at the base. The operation of iridotasis is still too young to receive its place in literature. The trephine operation is on the whole in bad repute, and Dr. Fuchs stated in his recent visit that he had performed about 1,000 trephine operations and that he had abandoned it on account of late complications. As to the operation of choice in greatly contracted fields, the trephine seems to be most popular, altho most all of these cases eventually go blind.

A Contribution as to the Etiology of Hydrophthalmos.

DR. C. A. CLAPP showed lantern slides of microscopic sections of a case of hydrophthalmos in a child dying at the age of 10 days with anencephalia. The eyes were greatly enlarged, there was edema of the corneal epithelium, thinning of the substantia propria with incomplete separation of the iris from the cornea, confirming Mr. Treacher Collins' observations.

Discussion. DR. JONAS FRIEDENWALD inquired if there was anything in the orbits to account for the exophthalmos.

DR. ZENTMAYER: My case, referred to by Dr. Clapp, had a coloboma of the iris but with a small root at the base. Microscopic study showed no canal of Schlemm.

DR. CLAPP: As far as one could determine nothing except the greatly enlarged eyeballs could account for the exophthalmos.

C. A. CLAPP, SEC.

SOCIÉTÉ D'OPHTALMOLOGIE DE PARIS.

February 17, 1923.

M. COUTELA, President.

Trophic Lesions of Cornea Caused by Unrecognized Foreign Body.

DR. M. BENOIT reported the case of a mechanic who had been injured ten years before in a boiler explosion, after which the left eye was under treatment for three weeks, but seemed to have recovered with full vision. Five years

later there were pains in the eye but vision remained good. Nine years after the accident the man, working with his head bent down, felt a veil before his left eye which remained when he applied for treatment, which did not relieve it. The cornea was irregular and clouded over its whole surface; but the pupil could be seen thru it, reacting normally. Fingers were counted at one meter. The eye became red and was sensitive to light. The cornea did not stain with fluorescein but showed marked anesthesia. The other eye was normal in all respects. Finally examination by strong daylight revealed a fragment of glass lying on the iris, in the angle of the anterior chamber, between six and eight o'clock; without doubt left there by the accident ten years before. The corneal microscope showed epithelial lesions with dull irregular surface and islets of healthy epithelium.

Pulsating Exophthalmos Treated by Ligation of Orbital Vein.

Drs. Rochon-Duvigneaud and Valiere-Vialeix presented a patient in whom there appeared, January 20, 1922, neuralgic pain in the right temporal region, lasting only three days; and a bruit heard in the right ear, which the patient compared to the beating of the heart. In March the eye became red and continued so. April 15th there appeared exophthalmos and marked diplopia on looking to the right, from paralysis of the oculomotor. Vision did not seem impaired. Treatment with mercurials was without result. In July the eye remained red, the exophthalmos had not increased, the diplopia continued, but less annoying. Distant vision was good altho the patient complained of difficulty in reading. Bruit and pulsation continued and were most complained of. When examined October 23 there was moderate right exophthalmos, somewhat downward but chiefly forward. The eye turned in, movements were slightly diminished up and down, and possibly outward. At the upper inner angle of the orbit was a prominence due to dilated vessels which pulsated, with the systole. Auscultation gave a systolic sound like a jet of steam, compression of the right carotid stopped the pulsation thrill, and sound. The vessels of the anterior segment of the eye were dilated. The tension was increased to 65 mm. Hg, with the tonometer. Pupil dilated, without reaction to light, anterior chamber shallow, vision, hand movements. Examination of nose and sinuses proved negative.

The upper and lower lids were united and two weeks later an incision was made in the upper part of the orbit. A greatly dilated vein, the size of a goose quill which bled like an artery, was exposed. It was secured by forceps which were removed after 18 hours. The improvement was positive but not complete. The swelling at the angle of the orbit disappeared, the exophthalmos diminished and the tension of the eyeball. Pain was completely relieved and the noise slightly diminished; but continuing to some extent. Attention was particularly called to the glaucoma present in this case.

Discussion. Dr. Morax reported a case of arteriovenous aneurysm from trauma in a girl of 19 years, from transverse compression of the skull. The internal carotid had been ligated, without great improvement. After 9 years there was still pulsating exophthalmos with convergent strabismus, 35 degrees. The dilated orbital and suborbital veins were exposed, without difficulty under local anesthesia, and ligated with catgut deep in the orbit. After 9 months the result remains very satisfactory, the exophthalmos was very slight, there was no pulsation or bruit.

Ionization in Ocular Therapeutics.

Drs. A. Cantonnet, G. Bourguignon and M. Joltrois, found this method of introducing substances into the organism neglected in ophthalmic therapeutics. The technic of cocainizing the eye and introducing the electrodes beneath the lids was disapproved. Retention of the corneal sensibility was necessary to avoid a harmful intensity of the current. The electrode should be applied to the eye thru an eyebath from which an open tube permitted the bubbles of gas to escape. The other

electrode was applied to the nape of the neck. A current of one-half to 1 ma. might be continued twenty to thirty minutes. It should be increased and decreased gradually. They used sodium iodid and salicylat, which should be applied thru the negative pole, and calcium chlorid to be applied thru the positive. The result of 1,500 treatments applied to over 80 patients were remarkably good in cutaneous scars, softening them and rendering the tissue supple, by use of sodium iodid.

The cure of external ocular palsies (by calcium chlorid) appeared to be hastened. For corneal opacities (sodium iodid) the results were very good—complete cure in recent cases and appreciable improvement in those of long standing. In iritis with synechiae (sodium salicylat or iodid) there was amelioration of pain and pericorneal injection. For scleritis (sodium salicylat) the action was variable. For optic atrophy (sodium iodid and calcium chlorid) the effect seemed clear on toxic amblyopias, and more or less marked for atrophy which might remain long stationary. Little or no effect was produced on choroiditis of myopia. With reference to cataract they were not prepared to give any conclusion. The procedure was inoffensive and painless.

Discussion. Dr. Morax had found a manifest action from zinc iontherapy in pneumococcus keratitis, apparently superior to that obtained by applications of zinc sulfat or tinctur of iodin.

Acute Retrobulbar Optic Neuritis, Polyp of Sphenoidal Sinus.

Drs. Velter and Liebault reported the case of a woman of 27 who came for impaired vision of the right eye of 12 days' standing. It began with deep pain in the orbit, increased by pressure and movements of the eyeball, without redness of the eye or swelling of the lids. Some days later the sight fell rapidly altho the pain grew less. The left eye was entirely normal. The pupil of the right was dilated with the complete loss of direct light reflex but preservation of the consensual reflex. Media clear, very slight hyperemia of papilla. Vision less than 1/100, field slightly retracted at the periphery, with very large central scotoma, absolute to 25 or 30 degrees. On examination the nose at first seemed normal; but on closer inspection justified operation on the middle turbinate, in the region of the sphenoidal opening. Forceps introduced into the sphenoidal sinus removed a polyp attached to the mucous membrane, and zinc chlorid 1 to 100 destroyed the debris. The next day the direct reaction of the pupil had returned and was more evident two days later, when the central scotoma had diminished and the vision slightly improved. At the end of ten days the vision was 1/25, the scotoma about 10 degrees in diameter, the peripheral field normal. At the end of a month V. equal 1/10, scotoma 3 or 4 degrees, and two weeks later V. equal 8/10.

Discussion. Dr. Morax could not share the view that the improvement in the symptoms was necessarily due to the removal of a small sphenoidal polyp. He had seen similar improvement without operative intervention or after opening of a maxillary sinus.

Dr. Velter thought improvement simply by coincidence very unlikely, as the return of the pupillary reflex was evident the next day.

Dr. Liebault pointed out it was easy to open the sphenoidal sinus if it were judged necessary. In this case there was very little secretion, but enough to examine.

Epidemic Encephalitis. Ocular Movements and Corneal Lesions.

Drs. J. Bollack and H. Lagrange reported the case of a man of 28, who seven years before had syphilis; who came with meningeal symptoms, fever, rigidity of neck, headache, some vomiting, intermittent crossed diplopia, Argyll Robertson symptom. Under injections of novarsenobenzol the symptoms improved and the temperature became normal. But at the end of a month there was bilateral ptosis, almost complete, spontaneous nystagmus, paralysis of vertical movements and convergence. The fundus and field of vision were normal. Vision reduced by corneal lesions. There was marked somnolence and indifference, slow speech, mask like face,

rigidity of head, neck and trunk. Wassermann reaction negative. There were superficial vesicular lesions of the cornea, vascularization of the middle and deeper layers, and keratitis with formation of epithelial threads. Inoculation of rabbits proved negative.

Exclusion of Lacrimal Sac for Pericystic Suppuration.

Dr. P. Toulant of Algiers thought, largely on account of trachoma, such lesions were frequent in Algeria among dispensary patients, who after cure of acute abscess generally refused ablation of the sac. Probing rarely gives good results, but exclusion of the sac by cauterization of the canaliculi and the lacrimal nasal canal was practised with very good results. After incision of the lacrimal abscess, washing out of the cavity, the canaliculi are dilated and the point of the galvanocautery introduced 3 or 4 mm. while cold, and then heated to a red heat. Then it is introduced in the same way into the upper end of the nasal canal and heated to a bright red. In six cases observed the cure of the abscess was very rapid; often obtained in less than a week. In only one case, incised before there was clear fluctuation, a second incision was needed followed by irrigation with Dakin's fluid. In two weeks there remained only slight redness, lacrimation diminished progressively. In these cases no recurrence was observed, in periods of four months to two years.

Retinocytoma without Rosettes.

Dr. J. Mawas read a paper upon this subject which will be published in translation with others closely related to it in a subsequent issue of this Journal.

Discussion. Dr. Morax had long believed that glioma of the retina (or retinocytoma) was a tumor in a class by itself, in that it always appeared at an early age, developed rapidly, was able to send off metastases, and had a special histologic structure. He pointed out it was in some respects similar to the tumors starting in the suprarenal capsule, both as to structure and clinical course. The suprarenal tumors are derived from cells of the sympathetic nervous system, and this might suggest the origin of retinocytoma from elements of the sympathetic contained in the retina. All known facts are necessary to determine our position with reference to the obscure question of glioma of the retina.

Dr. Mawas in reply pointed out that the tumors referred to by Dr. Morax were quite different from retinocytoma, which seemed more closely allied to certain tumors of the ependyma or the ventricular cavities. He would take up the differences between retinocytoma and other neuroepitheliomas at a subsequent meeting.

Optic Atrophy and Antityphoid Vaccination.

Dr. F. Terrien referred to the occurrence of lesions of the anterior segment following antityphoid vaccination. He had seen two cases of corneal ulceration. Bilateral iridocyclitis had also been observed. A reaction in the sheaths of the optic nerve might explain retrobulbar neuritis and true optic neuritis.

He reported in detail the case of a man, aged 43, who presented bilateral atrophy of the optic nerve. The vision was less than 1/40 for each eye and the visual fields greatly contracted. The pupils were moderately dilated and reacted poorly to light. The ophthalmoscope showed optic atrophy on both sides, apparently postneuritic. Fifteen minutes after antityphoid vaccination he presented ocular symptoms and in an hour could see nothing, and this continued four hours. Then he became able to distinguish objects and vision returned little by little, but very incompletely. At one time it reached 4/10, as reported by Bailliart, but afterwards again declined. The patient had a negative Wassermann reaction and there was no sign of tabes.

Discussion. Dr. Morax thought the case might be explained as resulting from a poorly prepared vaccine, or that coincidence might be invoked as the connection between the vaccination and the visual trouble. He inclined to the latter supposition.

Evulsion of Right Optic Nerve with Left Temporal Hemianopsia.

DR. DE ST. MARTIN, Toulouse. A young woman fell on her face before a reaper and, with other injuries, suffered evulsion of the right eye and optic nerve. The eye hung on the cheek, all the muscles being torn except the external rectus. The eye was enucleated after 48 hours, and next day the patient complained of left temporal hemianopsia. This remained complete, but with preservation of macular vision, after six months. V. equal 4/10. The whole optic nerve had been torn away. Among three cases of the kind in the literature, one had suffered temporal hemianopsia. Altho it could not be confirmed by anatomic examination or a radiogram, it was to be supposed that a part of the chiasm had been torn away with the right nerve.

Hypertension Following Corneal Puncture.

DR. A. MAGITOT has experimented on the influence of drugs upon the subsequent increased intraocular tension that follows tapping of the cornea. He finds atropin retards this hypertonic reaction. Pilocarpin and eserin also retard it, but less than atropin. Adrenalin appears to cause an increased reaction. He noted that the anterior chamber always reforms at the same time, whether a collyrium is used or not, and that instillation of either of the drugs mentioned prolongs the time during which the eye remains soft.

Vision of Drivers of Automobiles.

The Commission upon this subject was composed of Morax, Dupuy Dutemps, R. Onfray and P. Petit. They point out the factor of prudence is important, and enables certain drivers to avoid accidents even with poor vision. The influence of color sense, too, needs consideration. They conclude that the visual acuity should be not less than 3/10 in one eye and 1/20 in the other, with corrected ametropia. Those blind of one eye are thus excluded. The visual fields of the two eyes should be approximately normal. Those suffering from hemianopsia, contraction of the peripheral field, or large scotomata must be excluded. All persons subject to diplopia should be eliminated, and also those who suffer from hemeralopia. The vision of automobile drivers should be examined at regular intervals and in connection with any accidents in which they have been involved.

Discussion. DR. COULOMB thought the conclusions were a little too severe with regard to persons having only one eye. He suggested statistics should be obtained regarding drivers with this disability, some of whom were very skilful chauffeurs. Was it proposed to discontinue the license of those with one eye to whom it had already been granted? Or simply to exclude future applicants? He also questioned if the same standard should be applied in civil as in military service.

SPECIAL REPORT.

THE BARRAQUER OPERATION AND VITREOUS CHANGES.

CAPT. ROBERT E. WRIGHT, I.M.S.

MADRAS, INDIA.

The following is practically an extract from the report of the Government Ophthalmic Hospital in Madras, India, for 1922. It discusses the results obtained with this operation, and points out the effects of disturbances of the vitreous.

In addition to the 170 cases quoted, 80 others were operated by Barraquer's method, making a series of 250: 118 were delivered with ease, 56 with difficulty. Failure to deliver 76. The capsule burst, while attempting to remove by Barraquer's method, in 36 of these. Success, 80.4%. Partial success, 8.8%. Partial failure, 8.0%. Failure, 2.4%. Untested, 1. Vitreous escaped in 9.2%. Of 124 cases examined at the time of discharge, vitreous opacities were present in 94 and absent in 30.

It may be seen at once that the results are very much worse than in the case of our ordinary capsulotomy

method, but this is hardly a fair comparison. A definite opinion on the value of any method of cataract operation cannot be formed on as small a series as this, and it was obvious that the later results tended to become better. One got the impression that phakoerisis is probably more valuable than any other method yet introduced for the removal of the lens in its capsule in selected cases, but there are a large number of cases in which the method is inapplicable. Barraquer's method will hardly give a sufficiently high percentage of "operative" successes to justify its adoption in the following types of cases: (a) A bulging eye. (b) A friable capsule. (c) An immature cataract with a normal suspensory ligament. (d) Morgagnian cataract.

Other more uncommon types, too, might be enumerated in which it would be infinitely wiser not to use the method; and, in so far as the "operative" and "immediate postoperative" results are concerned, it is still beyond doubt that the surgeon must select his operation according to the case and not allow himself to be tied down to any particular method, even tho he prefers a certain type of operation for the general run of his cases.

In our experience one of the most important pitfalls into which the beginner is liable to stumble is allowing the cup to become engaged on the iris. It is absolutely essential that the pupil should be fully dilated, or an iridectomy be done if the operator has any difficulty in applying the cup to the anterior capsule cleanly. We have frequently found difficulty in keeping the cup clear of the pupillary border either in cases where full dilatation was obtained or in cases where the iris contracted as the result of making the section. It may here be mentioned that many of our cases dilate to a very limited extent with the usual preoperative instillation of atropin 1 per cent. The reason is not often obvious and such cases are inclined to resist 3 or 4 instillations. This type of case is not suitable for Barraquer's operation with iridectomy.

When with Barraquer's method a perfect delivery is effected thru a non-injured pupil and a negative pressure is established subsequent to delivery, with complete and perfect reposition of the pupil, one may consider that the most perfect operative result has been achieved in so far as the intracapsular method is concerned, provided of course that the "immediate postoperative" period is gone thru without mishap, that is to say, that the section heals, the chamber forms, and we get a round active pupil.

The above observations have altogether referred to the "operative" and "immediate postoperative" result. I have, for a long time, held that the greatest and most far reaching difference between any intracapsular method and the capsulotomy method (other things being equal), is that the vitreous body is deprived of the support of the suspensory and capsule, a partition which not only serves to retain the vitreous in position and allows the posterior chamber to form, but which prevents to a large extent invasion of the vitreous by cell elements which give rise to vitreous opacities. The remote changes to which the vitreous is liable as the result of this deprivation is the test by which the intracapsular method must eventually stand or fall, in the cases in which it is admittedly the operation of choice from a purely technical point of view. The great drawback about the formation of opacities in the vitreous and interference with vision thereby as compared with the formation of capsular opacities (after cataract) is, that after cataract is compatible with a healthy posterior segment, and may, with care, be relieved, whereas vitreous opacities of an organized type tend to be progressive and incurable.

For some time now I have been collecting evidence on this point and I may say, without presenting cases in detail, that I have formed the opinion that very few cases in which an apparently perfect intracapsular operation is performed, are free from invasion of the vitreous. I have no doubt that we, here in Madras, deal with a type of case which is very much more liable to reaction than is met with for

instance in Europe or America, and for this reason one cannot form a true opinion as to the ultimate results of leaving the vitreous unsupported by the suspensory capsule diaphragm. I have seen records of cases published in ophthalmic literature in which, with an "expression" operation, there has been 6/5 vision for many years with the appropriate correction; but in spite of this, I am almost persuaded that in so far as our cases are concerned, where no selection is made and constitutional disease does not constitute a contraindication to operation, it must frequently happen that the vitreous, left exposed with the very delicate (and sometimes torn or nonexistant) hyaloid to protect it, is invaded by cellular elements thrown into activity by the trauma of operation.

In investigating this hypothesis I have already made a considerable number of examinations with the Gullstrand slit lamp and corneal microscope after the method of Prof. Vogt, and have observed that even in perfect "operative" and "immediate postoperative" results with Barraquer's technic (or any other intracapsular technic) examination at the time of discharge (14 to 21 days after operation) almost invariably reveals masses of pigment not only on the surface of the vitreous but extending back into the substance of the vitreous. In many instances it is impossible to say whether these masses of pigment represent migrating melanoblasts or merely tiny deposits of actual pigment, but in certain cases it would appear that cells are undoubtedly migrating and such cells are capable of giving rise to delicate fibrillar networks which radiate backwards thru the vitreous body. The manner in which the vitreous responds to traumatism unassociated with bacterial invasion need not be gone into fully here.

My general impression from the microscopic observations on numbers of specimens is, that the vitreous, once invaded by cellular elements, is very liable to eventual opacities of a serious nature, and that opacities of this type once formed are unalterable. One does not contend that there is no such th'ng as amorphous vitreous opacities capable of undergoing absorption either spontaneously or in association with some form of treatmert, but the possibilities referred to here do not come under this head. The type of vitreous change which is referred to is that associated with trauma and with the possibility of cells being set free into the vitreous, either from the surface of the retina as might occur in the bursting of the vessel, or the penetration by a foreign body which carries cellular elements in its track, or the performance of an operation which removes the smooth posterior capsule and suspensory from the vitreous in front and allows the cellular elements of the iris and traumatized ciliary to invade the vitreous itself. The hyaloid evidently is frequently ruptured even in the most favorable cases, altho one has frequently been able to demonstrate the fossa patellaris by means of the irrigator after a favorable intracapsular operation. The rupture of the hyaloid is sometimes evidenced by the fact of a mushroom like mass of jelly protruding thru the pupillary aperture covered with old dust like pigment spots and permeated by fine opaque fibrillar strands. At times one sees a formed posterior chamber with an apparently intact hyaloid covered with fine gold dust like pigment points.

It seems reasonable enough to suppose that the main mass of the vitreous is merely extracellular material derived from cell bodies for the most part situated in the region of the ora; and that the actual hyaloid membrane itself does not contain cells, but is a derivative of cells so situated; which throw out long cellular processes of a very delicate nature, which hold a semifluid matrix in their interstices. These, in different position, give rise to different appearances, such as the fibers which run forward to become continuous with or form the suspensory itself, and the fibrils which welded together dip behind the lens to form the delicate homogeneous hyaloid and line the patellar fossa. It must not be forgotten that in traumatizing the suspensory we are probably traumatizing that region from which the solid ele-

ments of the vitreous itself spring. The suspensory is for all practical purposes identical in origin with the enveloping membrane and radiating transparent solid fibrillar portions of the vitreous mass.

It is perhaps impossible in the present state of our knowledge to fully understand why the vitreous fibrils should become opaque when a trauma is effected by a perfectly sterile foreign body. Loss of fluid with shrinkage is partially responsible, by giving rise to altered conditions of refraction which enable the solid elements of the vitreous to reflect light. Round cell migration (infiltration) is another condition easily recognized in specimens, but this change is only met with in gross and obvious disease. A third variety of vitreous change is that which depends on invasion by delicate connective tissue elements. The relation between the transparent fibrillar supporting structure and interfibrillar fluid is upset, the solid elements become opaque, the fluid altered in physical qualities. Sir William Lister's address at the Washington Congress sets forth views practically in line with our experience and observations, almost the only divergence being the distribution and significance of the distribution of recent vitreous hemorrhages. One need not say anything further to draw the attention of those who are interested in this matter to the great importance of treating the vitreous with respect in all operative procedures on the eye. In this clinic we regard an eye as potentially lost in which a vitreous escape of any kind has occurred, or in which vitreous comes to lie against the back of the section, or even when there is a hammock shaped, drawnup pupil, with the pillars rolled backwards and the vitreous occupying a plane anterior to the iris in the upper third of the anterior chamber. We would like to draw the attention of those who appear to regard vitreous trauma of various kinds as a mere incident, undesirable in nature, but of no great importance as regards eventual prognosis, to the importance of a prolonged and accurate study of the changes which take place in the vitreous as the result of development of fibrillar streamers from the region of the trauma.

The eventual test of such vitreous changes is, of course, the vision and it must be confessed that many cases, in which there is a considerable vitreous change on microscopic investigation, preserve a fairly good vision for a considerable time. Strictly speaking the result of an operation for cataract ought to be recorded at least 18 months after section. In this way only, will the truth be arrived at as to the permanent and lasting value of any method. This is a very difficult matter in this country, but no difficulty ought to be experienced in gradually collecting large figures in other countries where hospital cases are more easily kept in view. If only the perfect "operative" and "immediate postoperative" results were followed up, the figures would be strictly comparable. I do not lose sight of the fact that it is better for a patient to have vitreous opacities and gradually darkening eye than not to have vision at all, but one must seek the truth. My contention may be quite wrong when applied to selected eyes, healthy except for cataract, such as are selected for operation in Western clinics. In time the accurate records of carefully followed up cases will show.

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OPHTHALMIC PATHOLOGY.

If every surgeon were compelled to study with the microscope each piece of tissue he removed from the human body, medical science and humanity would gain greatly. The specialist who devotes himself to the study of pathology was needed, and has come to stay. But he must meet the danger common to all who confine their work to one branch of medical science; the danger of losing touch with other branches, and becoming on that account less useful and less intelligent, even in the special field he has chosen.

The facts of pathology take significance so largely from the related facts of clinical medicine, that to lose sight of their interrelations is to miss their broad significance. The danger of doing this is especially great for any worker in the laboratory, who lost touch with clinical medicine when he left the medical school; and now sees pathology only as it is shown in the isolated specimen. The narrowness of purely laboratory workers has been peculiarly detrimental in ophthalmology, because ocular pathology depends so much upon the minute structure and relations of the parts.

Then so much of ocular pathology is studied with the ophthalmoscope, the corneal microscope, the orthoscope and the transilluminator, that the laboratory worker, unfamiliar with these, has a very incomplete knowledge of his specialty, when he comes to pronounce upon the section he finds under his microscope from an enucleated eye. Opaque media, new formed tissues, isolated cells, or exudates in the eye, are not peculiar in their shapes or staining, but in the effects produced by their location; and in the sequence shown by the pathologic changes.

A carcinomatous mass or an inflamed appendix may carry the essentials of its story in its microscopic structure; but an enucleated eye will lose much of its interest if not fixed, cut and stained with reference to points suggested by its clinical history and evidence afforded by its study during life. The giant cells of sympathetic ophthalmia, tuberculosis and those surrounding cholesterin crystals may look alike under the microscope; but have totally different meanings when considered in the light of the clinical history of the eye that is being examined. Changes in the ciliary epithelium, or

the connective tissue adjoining Schlemm's canal, might mean nothing to one who had never anxiously observed the changes of intraocular tension, or alterations of the visual field.

Professor Leidy used to say to his classes in anatomy: "What will be seen with a microscope does not depend so much on what is under the microscope as on what is at the upper end". Any one can look thru a microscope and see whether a cell is round, or oval, or spindle shaped, or stellate. But that does not make him a pathologist. No perfection of technic, thinness of section, or perfection of staining, alone, will enable a laboratory worker to give a valuable opinion on the significance of changes visible in an enucleated eye. It is the knowledge of the significance of what is visible, the appreciation of the pathologic relations of the appearances observed, that gives value to a judgment based on what is seen thru the microscope.

To give the best assistance to the ophthalmologist, the pathologist must know a good deal of the diseases of the eye. He should be familiar with ophthalmo-pathology, as revealed by the ophthalmoscope and corneal microscope. He must know the general course and interdependence of ocular diseases. But to most general pathologists, these are unexplored branches of medical knowledge. Real proficiency in them is only found among those who have studied and practiced ophthalmology, and who to their ophthalmic training have added months and years of careful work in well conducted laboratories of pathology. If an ophthalmologist wishes good assistance as to the pathology of an eye he has enucleated, he should seek the assistance of one of these ophthalmo-pathologists.

This is no merely academic, or theoretic matter. It is generally true that the ophthalmologist, who relies on the general pathologist for help, gets very little and very unsatisfactory assistance. If he is not actually misled by a wrong diagnosis, as sarcoma for "glioma of the retina," he still does not get from the enucleated eye that positive and basic interpretation of the changes he has observed during life, that he has a right to expect. Post-mortem examination ought to verify, or correct antemortem diagnosis, in all its ramifications and refinements; and this the report of a general pathologist on an eyeball, sent to him, very rarely does. Of course, it is possible for a pathologist to study the minute anatomy and diseases of the eye, until he is competent to give this help. It is also possible for the ophthalmologist to study pathology in the same way, so that he can help himself. But very few will, or can do either of these things, to work out an individual case.

The practical thing for most of us, is to send our enucleated eyes to an ophthalmo-pathologist. These colleagues are, without exception, keenly interested in the advancement of ophthalmic science, glad to get new material for their investigations, and ready to give really valuable reports to their ophthalmic colleagues, based on the work of thoroly competent assistants checked by their own observations, for fees absurdly small, in comparison with the time consumed, or the value of the results. Any semblance of excuse for not having a careful pathologic examination made of every enucleated eyeball, is removed by the opportunity afforded by the co-operation of the United States Army Medical Museum with the American Academy of Ophthalmology and Oto-Laryngology. Every such specimen, sent with a proper history of the case and properly preserved, will be skillfully cut and stained, studied by a competent committee of ophthalmo-pathologists and a description and specimen section sent to the donor.

Ophthalmic pathology is already far in advance of most branches of pathology. Its further, rapid development is within easy reach of the readers of this journal. Let us give a little time and thought to this matter. Few things we can do will bring such a rich return to ourselves, our patients and our successors.

E. J.

SPECIALISTS TRUSTEES IN THE A. M. A.

At the foot of page 1012 of the December issue of this Journal occurs a sentence that misrepresents the present policy of the American Medical Association towards specialists, and calls for correction. It states that the A. M. A. never had a specialist on its Board of Trustees. The fact is, that of nine present members of that Board, four have practised chiefly ophthalmology or oto-laryngology and one is a neurologist and psychiatrist.

These men have not been chosen to this responsible office because they were specialists; but because they were men of the highest standing in the profession, and known to possess the business experience and judgment needed to conduct and safeguard the large undertakings and property interests of the Association. After their fitness had been recognized by the House of Delegates, to which most of them had been elected by their State Medical Societies, they were chosen to administer this great trust. The fact that they were specialists, and not general practitioners or surgeons, was no bar to such advancement.

The American Medical Association has reflected the general attitude of the medical profession in America towards specialists, as it has with regard to other matters. A generation ago when specialism was new that attitude was one of skepticism and distrust. To claim special skill in a particular class of diseases was an old device of the quack, who knew nothing of medicine in general. The profession had to be shown that special study and thought on a limited branch of medicine would make the specialist a better consultant and a better advisor for the public. This has been shown, and the general profession has accepted the proof. The time has come to forget that formerly it was otherwise. A little more promptness of forgetting what is no longer true, a little more care to correct preconceived notions by a reexamination of facts, would have prevented the writing of such a misleading sentence. E. J.

EDITORIALS.

The brief, impartial, impersonal summary of a subject from a basis broader than the experience of the writer, but giving views arising from his own study and thinking has an important place in medical literature. Without thinking that he has anything wholly new, that would justify a long formal paper for its presentation, one may feel that he has gained from his own experience an idea regarding some important point in ophthalmic practice, that has not become current, that is not taught in the text books, or easily discovered in the periodic literature of ophthalmology. To present such ideas the editorial form is especially adapted.

There are matters of temporary interest, or concerning the professional rather than the scientific side of ophthalmology, that can well be noticed in editorials. But the most important, most widely useful editorials, for an ophthalmic journal, deal with points bearing on ophthalmic practice. The number of these having a full measure of such importance and usefulness that can be expected from any one writer—even the most fertile and experienced—is quite limited. But the number of ophthalmologists who might furnish one, or a few editorials of this kind, is large. It is hoped that the editorial management of the A. J. O., will be able, from time to time, to interest new contributors to this department, who will give permanent form to practical points in diagnosis and therapeutics, by which they and their patients have frequently profited.

The presumption is, that what has proved its value in the practice of one will be of equal value in the practice of others; and to most readers the brief, direct, didactic form of the editorial will be found the best form of presenting it.

Generally the attempt to give definite expression to one's ideas will be found, better than anything else, to clear them up, to show their limitations, their connections with other things, their wider applications that had not before been noticed. A proper sense of the responsibility which

should attach to putting things in print, will cause the writer to look into the literature, to find if his thought has been anticipated and when or by whom. Sometimes a most valuable service may be rendered by calling attention to a suggestion of value, long overlooked or forgotten.

When an editorial has been written it may well be kept for a few weeks and looked over again and again; to make sure that it is as brief and as clear as possible; and that it puts just the right emphasis on the main idea, without undue repetition or sweeping statements that are liable to excite opposition in the mind of the reader.

On the other hand, to illustrate the better way, some of the editorials on points of practice have been under consideration for weeks. Hours or even days have been spent in looking over the literature relating to the subject; not to cite authorities, but to make sure that the statements made are in accord with all the known facts, and that their implications are equally well supported. If one will concentrate on the writing of two pages, the time, thought and study that is often devoted to twenty pages, his time will be better spent, his thought will reach more readers, and will make a deeper impression on the minds of his readers. It will be more advantageous to his reputation as a writer and more beneficial to his readers.

Let no reader hesitate to try what he can do in this direction. Any good talker can become a fair writer if he will do some real work in the direction of writing clear, short sentences, choosing direct expressions, avoiding repetition, eliminating thoughts that may be good in themselves but have no essential relation to the main thought that he is trying to express, trying always to find the best word for his purpose, among several that mean nearly the same thing.

The better medical journals today draw their editorials from a wide staff of writers. It would be mutually helpful if many readers would undertake this particular kind of writing for this journal. To get new points of view on subjects much thought about, is more helpful than anything else that reading can give us; and the effort of writing is always worth while, to clear and focus one's thinking. The department headed "Correspondence" might well be similar in character to the Editorials. Some who will not feel like attempting editorial writing will undertake a letter to the editor, and that department would be better for their contributions. Whatever help the editor can give, will be freely at the command of those who seriously try to do good work in this direction.

E. J.

MR. SYDNEY STEPHENSON.

The death of this leading British ophthalmologist, on December 15th, 1923, has just come to our notice. In 1903 he established the "OPHTHALMOSCOPE," and made it at once one of the most important ophthalmic journals in the English language. This position was maintained under his management until, in 1917, it was merged with the ROYAL LONDON OPHTHALMIC HOSPITAL REPORTS and the OPHTHALMIC REVIEW to form the BRITISH JOURNAL OF OPHTHALMOLOGY, of which he was Editor to the time of his death.

He made many friends in America, during his visit to this country in 1910, who will feel his death as a deep personal loss. Able successors will carry on the work to which he devoted his best energy; but all ophthalmologists will regret that our literature will have no further contributions from his pen. More adequate notice of his work will appear later.

E. J.

CORRESPONDENCE.

The V Test for Astigmatism.

To the Editor: I learned with great interest from Dr. Verhoeff's article in your number for November, 1923, that my V test had been anticipated by him in 1899 with his chart of two cross lines at right angles. I have not been able to obtain a copy of the *Ophthalmic Record* for that year (see below); but will say that I have, for so many years, admired the ingenious mind of Verhoeff, as to consider it an honor to be anticipated by him, even in so small a matter. I found the V test so useful for so many years as a pointer, and then as a confirmer, that I thought it worth communicating, tho it would be strange if so simple a principle had been new. The angle I mentioned to the manufacturer (after experiment) was 45 degrees, but Verhoeff's cross lines, at 90 degrees are the essence of simplicity, since they can be used for both axis and the amount.

Congratulations to Dr. Verhoeff.

Yours truly,
E. E. Maddox.

Bournemouth, England.

E. E. Maddox, Esq., M.D.,
Bournemouth.
Dear Sir:

In reply to your letter I am sorry to say the volume of the *Ophthalmic Record* for 1899 was missing from the set when the ophthalmological library was brought here in 1912, and we have not been able to replace it. If you could let me know which article in it you require I might possibly find a reprint of it in one of the other journals.

Yours faithfully,
H. E. Powell,
Librarian.

Royal Society of
Medicine, London.

Ophthalmologists in Germany.

To the Editor: Lately I came across a couple of numbers of your AMERICAN JOURNAL OF OPHTHALMOLOGY, which I read with the utmost of pleasure and a great deal of profit. I should be glad if I could continue, but, alas, for obvious reasons I am not able to pay the subscription. Would it be too immodest to ask you to let me have every now and then, gratuitously, a spare number?

You would greatly oblige me if you would be so kind to let me know the fees of American oculists. The local association of physicians has resolved that we have to charge Americans from three to five dollars for consultation. Most of them pay willingly, but some reluctantly. Do repectable eye specialists furnish spectacles? I was expected to do it when I charged seven dollars for a very long and tiresome consultation (astigmatism combined with insufficiency) in a very nervous person.

———. ———.

Dresden, Saxony, Nov. 10, 1923.

Tonometry. A Correction and Apology.

To the Editor: In the American Journal of Ophthalmology for September, 1920, was published a paper of mine with the title, Tonometry and the Prevention of Glaucoma. In that paper I assumed that a water manometer should be calibrated on the assumption that the column of water should be eleven and one-ninth times the height of mercury. From the fact that none of my readers called attention to my error, I suspect that they were as ignorant of the true multiplier as myself. Some months ago Dean E. P. Lyon of the University of Minnesota told me that the atomic weights, 200 and 18 were true only of mercury and water in the gaseous condition, and that my multiplier should have been thirteen and six-tenths. If my error had been pointed out earlier I would have been saved much perplexity. My first tonometer was based on a mercury manometer, and when for greater convenience and accuracy I constructed a better artificial cornea and, as I flattered myself, a more accurate (water) manometer, I found myself involved in difficulties that I was unable to solve. The later change of my water manometer to a correct graduation removed most of the trouble and gave me comparative measurements which were more favorable and more just to the older tonometers. This paper, then, is an apology as well as correction. I

have delayed writing this in the hope of being able to publish corrected figures for all the instruments on the market. I hope, sooner or later, to be able to do this, but at present am able only to give the comparative measurements of the Schiötz, McLean and Brown tonometers. Dr. Gradle and Dr. McLean have both questioned the validity of the artificial cornea as compared with the human cornea as a basis for measurements. But I am compelled to believe that, taking into consideration the varying radii of curvature of human corneas, as well as the numerous other factors involved in intraocular tension, the artificial cornea affords a more constant and a safer basis for calibration of the tonometer than any human or animal eye. Dr. Gradle has given me most cordial cooperation in this work. In experimenting with artificial corneas I found that the two or three layers of rubber dam stretched too much with the higher pressures. This was improved upon by using one thickness of dam covered with a layer of very thin old linen. Dr. Charles Nelson Spratt suggested two layers of gold-beater-skin over the rubber, and I have found that fairly satisfactory. I make the artificial cornea as follows:

ARTIFICIAL CORNEA.

Draw a rubber finger cot lightly over the end of a tube of 12. mm. diameter, pour in water to the equivalent of 30 mm. of mercury, then adjust carefully two thicknesses of gold-beater-skin and tie tightly.

These figures are the result of a large number of measurements (this is the final series) in which I took infinite pains to be more than fair to the older instruments, taking practically the highest readings in the older as against the minimum readings of the Brown tonometer.

COMMENTS.

The fact that the Brown tonometer gives readings more closely consistent with the manometer is to be expected because it was calibrated on that basis. It does not prove that the instrument is better or worse than the older ones.

The measurements I have given do, however, suggest to me very strongly that if men who are fairly competent to use the various tonometers and have taken infinite pains to make fair and accurate comparisons get such conflicting results, tonometric measurements by the rank and file of ophthalmologists are open to a great deal of suspicion. These results seem also to indicate that it is both desirable and possible to standardize tonometers and tonometry. To that end, I am confident that my six millimeter foot-plate is a contribution, and that it is decidedly superior to the ten millimeter foot-plate of the Schiötz and McLean, since it eliminates a considerable number of intangible influences which cannot be estimated in any particular case.

EDWARD J. BROWN, M.D.
Minneapolis, Minn.

COMPARATIVE MEASUREMENTS.

	Schiötz Tonometer					
Manometer	15 Gr.	10 Gr.	7.5 Gr.	5.5 Gr.	McLean	Brown
100	92	87	62	46	95	95
90	83	70	62	46	80	90
80	72	83	62	46	65	75
70	54	52	46	52	70	65
60	30	50	43	46	70	60
50	25	32	37	46	50	50
40	22	27	37	30	30	22
30	..	20	26	25	20-25	22
20	..	14	18	21	15	18
15	..	..	12	17	12	15
10	..	..	..	15	..	10

Coronary Cataract and Agricultural Conjunctivitis.

To the Editor: I would like to make a slight correction with reference to the article by Dr. Cassidy and myself appearing in the September number of the JOURNAL. We stated there, speak-

ing of coronary cataract, that this "according to Vogt is never progressive and never interferes with vision." During the course at Zurich I found that Vogt has seen this condition gradually progress far enough to interfere considerably with central vision, the opacities gradually becoming thicker in the central area of the posterior cortex, altho the condition by itself never goes on to complete maturity in the ordinary way. On referring back to Vogt's atlas I find it mentioned also that the opacities may in some cases interfere with central vision.

I would like also to add a note of explanation regarding the case of agricultural conjunctivitis reported on page 764 of the same number. The report was sent in by mistake without this part of it. The case was reported on account of its almost exact resemblance to a series of cases reported by Dr. Patton and myself in the AMERICAN JOURNAL OF OPHTHALMOLOGY for 1922, which we called "Agricultural Conjunctivitis" because they appeared chiefly in farmers. These cases were characterized by the cardinal symptoms of superficial necrosis of the skin, swelling of the preauricular glands, and membrane formation on the conjunctiva. These symptoms were all present in this case; and likewise the large gram positive bacillus, found in the smears but not growing in aerobic cultures, seemed to be the same as that found in several of our previous cases.

Sincerely yours,

Omaha, Neb. S. R. GIFFORD.

ABSTRACT DEPARTMENT

Reprints and journal articles to be abstracted should be sent to Dr. Lawrence T. Post, 520 Metropolitan Building, St. Louis, Mo. Only important papers will be used in this department, others of interest will be noticed in the Ophthalmic Year Book.

Brownlie, W. Implantation Cyst of Iris and Ciliary Body After Advancement Operation With Pathologic Report by **H. Neame.** Brit. J. Ophth., v. VII, 1923, p. 497.

Scleral cysts and implantation cysts of the cornea and ciliary body following injuries have been recorded, but the author was unable to find, in literature, a single case where the iris and ciliary body alone were involved after an advancement operation.

A girl, aged ten years, on inspection, presented a cyst of the iris occupying the lower temporal quadrant. The anterior wall was flush with the back of the cornea. The walls were transparent, the posterior appearing bluish-black. The adjacent conjunctiva presented a striated apearance, with two small round black spots, such as are observed following an advancement of the external rectus. The parent admitted that an advancement operation had been performed, ten months before. Vision = 6/60. As the child suffered pain, removal of the cyst was advised. With a Graefe knife a limbal puncture was made but as the cyst wall was in contact with the cornea it collapsed. A broad iridectomy tearing the iris from its base was done. Two months later a recurrence was noticed. Six months later, at the solicitation of the parent, a second attempt at removal was made, thru a trephine opening (2.5mm) at the site of one of the black spots in the conjunctiva. The cyst wall and contents were removed. The scleral opening soon became blocked and a second recurrence presented. The eyeball was removed three and a half months later.

The globe was sectioned and a microscopic report of the cyst made. The cyst presented several types of epithelial cells.

It was impossible to determine the exact site of origin owing to the disturbance due to successive operations. Implantation of epithelial cells in Schlemm's canal, or pectinate ligament, might possibly result in the formation of such a cyst.

The author observes that the sclera in a child, being thin, is easily perforated with the needle; but what is more important than too deep a bite, is to avoid carrying a tag of conjunctiva along with the suture into the needle hole. Seven illustrations accompany the contribution. D. F. H.

Morax, V. and **Lagrange, H. Choked Disc Caused by Tumor of Aqueduct of Sylvius.** Ann. d'Ocul. 1923, v. 160, p. 445-456.

The patient complained of lacrimation and a slight cloud in front of his eyes. The left lacrimal passageways were impermeable, but the right were normal. The vision, visual fields and pupillary reactions were normal, but the ophthalmoscope showed a high degree of choked disc. A lumbar puncture showed nothing especially abnormal, but was followed in six hours by headache and difficulty in speaking. The next day, there was hemiplegia and aphasia, and the patient died nine days later. The autopsy showed a hematoma of the size of a man's hand on the left side, involving the region of the fissure of Rolando and the left frontal lobe. There was also a reddish-white tumor occupying the aqueduct of Sylvius and extending forward to the anterior corpora quadrigemina. Illustrations show the position of the tumor. Microscopic examination of the latter showed it to be a neurocytoma of epithelial nature, with unequal distribution of rosettes. C. L.

Holmes, G. Optic Neuritis in Relation to Cerebral Tumors. The Med. Press, Dec. 5, 1923.

Holmes affirms that papilledema is unquestionably the most valuable sign when it is present in these cases. Unfortunately it is frequently absent, especially in the early stages of the illness. It is rare when the patient first seeks relief, for headache or other symptoms. It is well marked in the last stages. Its occurrence also depends a good deal on the situation of the tumor: in growths involving the pons it is rare, while in cerebellar tumors it is almost invariably present. In deep seated growths involving the basal ganglia, it rarely becomes manifest; in tumors of the midgrain it is practically always an early sign.

Other retinal changes may be easily confused with papilledema; for example, the neuroretinitis occurring in renal and arterial disease. The differential diagnosis is of great importance, inasmuch as both in renal and arterial diseases the retinal signs are often associated with headache and occasionally with symptoms of local cerebral disease, thus adding to the difficulty of a correct diagnosis. The congenital anomaly of the optic disc, known as pseudopapillitis, which is generally associated with a high degree of ametropia, is sometimes difficult to distinguish from true optic neuritis. The chief point in the differential diagnosis is that renal and arteriosclerotic changes are limited to the disc and its immediate neighborhood, while in papilledema the earliest and most essential feature is congestion of the retinal veins, and vision, despite the amount of the swelling, is not seriously affected until some degree of secondary atrophy supervenes. The term cerebral tumor he defines as including not merely new growths, but also cysts, chronic abscesses, local collections of fluid, etc.

H. V. W.

Valude and **Chantier. Optic Neuritis. Trephining of Sphenoidal Sinus.** Ann. d'Ocul. 1923, v. 160, p. 649-651.

A patient, who had suffered from frequent headaches, suddenly presented injection of the left eye, severe periorbital pain and pasting together of the lids. V =1. About three weeks later, vision was reduced to perception of light, while the ball was normal externally. Examination showed a mild hyperemia of the disc. Altho it was not possible to use the perimeter, the visual field taken with the hand seemed normal, with no scotoma. The hyperemia increased slightly, with some veiling of the margins. All etiologic examinations were negative, and treatment with mercury and salvarsan had no effect. It was finally decided to open the sphenoids. The right was small; the left large, with a slightly cloudy mucosa. The headaches at once disappeared and the vision gradually returned to normal.

C. L.

Terson, A. Orbital Optic Neuritis with Bilateral Total Blindness. Ann. d'Ocul. 1923, v. 160, p. 345-347.

The patient, aged 41, in January,

1922, had an attack of grippe, which was followed by intense temporo-frontal pains. On March 23, he complained that his right eye had become blind in 24 hours. The left eye became blind shortly afterwards. Externally, both eyes were normal except for immobile, slightly dilated pupils. Ophthalmoscopy showed a mild degree of optic neuritis. Local treatment with sweating, diuresis and light diet was followed in 13 days by return of vision, first in the right eye. This was gradual, lasting several weeks. The discs were whitish-yellow and slightly excavated. C. L.

Salvati. Instillation of Antipneumococcic Serum in Serpiginous Ulcers of the Cornea. Ann. d'Ocul. 1923, v. 160, p. 570-571.

Three cases are reported. In the first two, all therapy was in vain until the serum was used, when a cure was obtained. In the third, immediate recourse was had to the serum and to excision of the lacrimal sac, and cure was obtained in about two weeks. C. L.

Kalt, E. Technic of Cataract Operation. Ann. d'Ocul. 1923, v. 160, p. 689-699.

The purpose of a cataract operation is not only to remove the contents of the capsule, but also to do this in such a way as to avoid a secondary cataract. Statistics are given of the last 205 operations, together with the author's technic.

Certain precautions are necessary: a transient slight mydriasis; use of a suture; and a peripheral iridectomy. These are briefly discussed, as well as the method of anesthetizing, fixing the ball and making the corneal section. The use of his special capsule forceps is described with more detail. He tries to detach a greater or less piece of the capsule, but if this is too thick he tries to extract the lens in the capsule by gentle traction aided by pressure of the fingers of the left hand upon the ball, transmitted thru the lower lid. Sometimes the lens ruptures, permitting the nucleus to come out, but leaving the capsule behind. This latter is extracted with his forceps. After the operation, atropin is used, only one eye covered, and the patient is put to bed for one day. The suture is removed on the fourth to sixth day. From his results, he believes that the removal of a large piece of the capsule gives the best ultimate vision. C. L.

Weill, G. Opening of the Capsule and Secondary Cataract. Ann. d'Ocul. 1923, v. 160, p. 700-705.

Two series of cataract extractions are discussed. The first consisted of 150 extractions with iridectomy and capsulotomy. In this series, it was necessary to do a secondary operation 75 times (50%), divided as follows: First year, 53 cases; second, 19 cases; after the second year, 3 cases. The second series consisted of 272 extractions by the use of the capsule forceps, and usually without iridectomy. Of these, 48 cases (17%) required a secondary operation as follows: First year, 34 cases; second, 7; after the second, 7. The author believes that secondary cataracts were a little more frequent in cases operated upon before maturity. From his statistics, he claims that the use of the cystitome should be replaced by the capsule forceps. C L.

Lagrange, H. Pneumococcic Panophthalmitis and Pneumococcemia. Ann. d'Ocul. 1923, v. 160, p. 626-629.

Three cases are reported. In the first, an attack of grippe was followed in five days by symptoms of right panophthalmia, requiring enucleation. An almost pure culture of pneumococci was obtained from the neighborhood of the globe, from the anterior chamber and from the blood. The second case was seen after perforation of an ulcer of the cornea. The pus from the perforation on culture showed pneumococci, staphylococci and a club shaped bacillus, while the blood gave a pure culture of pneumococci. The third case followed a trauma with intraocular penetration of a foreign body. Examination showed gram positive diplococci in the intraocular pus and a pure culture of pneumococci from the blood. C. L.

NEWS ITEMS

Personals and items of interest should be sent to Dr. Melville Black, 424 Metropolitan Building, Denver, Colorado. They should be sent in by the 25th of the month. The following gentlemen have consented to supply news from their respective sections: Dr. Edmond E. Blaauw, Buffalo; Dr. H. Alexander Brown, San Francisco; Dr. V. A. Chapman, Milwaukee; Dr. Robert Fagin, Memphis; Dr. M. Feingold, New Orleans; Dr. Wm. F. Hardy, St. Louis; Dr. Geo. F. Keiper, LaFayette, Indiana; Dr. George H. Kress, Los Angeles; Dr. W. H. Lowell, Boston; Dr. Pacheco Luna, Guatemala City, Central America; Dr. Wm. R. Murray, Minneapolis; Dr. G. Oram Ring, Philadelphia; Dr. Chas. P. Small, Chicago; Dr. John E. Virden, New York City; Dr. John O. McReynolds, Dallas, Texas; Dr. Edward F. Parker, Charleston, S. C.; Dr. Joseph L. McCool, Portland, Oregon; Dr. Richard C. Smith, Superior, Wis.; Dr. J. W. Kimberlin, Kansas City, Mo.; Dr. G. McD. Van Poole, Honolulu; Dr. E. B. Cayce, Nashville, Tenn.; Dr. Gaylord C. Hall, Louisville, Ky.; Dr. Edward D. LeCompte, Salt Lake City.

DEATHS.

Dr. James F. Bowman, New York City, aged 44, died Nov. 28th.

Dr. William L. Friedman, Oakland, Calif., aged 51, died Dec. 1.

Dr. Wm. M. Reid, Kansas City, aged 57, died Nov. 2, of heart disease and influenza.

Dr. G. L. Mitchell, formerly of Plymouth, England, died Oct. 2, 1923, aged 29 years.

The veteran ophthalmologist of England, T. Pridgin Teale, died Nov. 13, at the age of 92 years.

Dr. Albert F. Mattice, Seattle, died Nov. 29, from a self inflicted gunshot wound.

Mr. Sydney Stephenson, editor of the British Journal of Ophthalmology, died Dec. 15, 1923, in London.

Dr. John William Stirling, of Montreal, died Dec. 10, after a month's illness, aged 65 years.

PERSONALS.

Dr. Charles Maghy has moved from San Francisco to Los Angeles.

Dr. Monod has been appointed chief of the ophthalmic clinic at Bordeaux.

Dr. W. D. Horner has returned from Vienna and opened offices in the Union Square Bldg., San Francisco.

Dr. L. Webster Fox and Mrs. Fox sailed on Saturday, Dec. 15, for Porto Rico, to spend the Christmas holidays.

Dr. Arthur J. Griot has just returned from Europe after seven months' study in Vienna and Budapest.

Dr. George A. Huber, formerly of Coffeyville, Kan., has opened offices in the Wagner Bldg., Santa Monica, Calif.

Drs. F. A. Aylesworth, C. C. Hill, S. M. Lyon and W. W. Wright have been appointed senior demonstrators in ophthalmology at the University of Toronto.

Dr. T. J. Williams of 30 N. Michigan Blvd., Chicago, has taken into association with him Dr. J. E. L. Keyes of Toronto, Canada, formerly of Edinburgh. Dr. J. D. Simons will also be associated with Dr. Williams.

Dr. and Mrs. William A. Fisher, of Chicago, sailed for India in December. Dr. Fisher expects to perform about a thousand cataract operations in Dr. Holland's clinic in Shikarpur during January and February.

Dr. Casey Wood has returned to this country after an absence of about eight months in the islands of the southern Pacific and, for a time, will be located in Berkeley, Calif. That the doctor makes the study of bird life not only his favorite pastime but a source of profit to others may be gathered from the following clipping published in "The Pacific Age," Suva, Fiji: "Owing to the hospitality of Doctor Casey Wood, the visiting ornithologist, the Suva community were afforded an opportunity of seeing the magnificent collection of the pictures of the birds of Fiji which he has made during his stay in the colony. * * * Work of the description carried on by Dr. Wood should be of the greatest educational interest and value in the colony, and we have no doubt that the Government will profit by the generosity and good nature of this gentleman, and obtain records which will be of very great use and interest.

SOCIETIES.

The annual meeting of the Illinois State Medical Society will be held at Springfield in May. Dr. Wm. R. Fringer, of Rockford, is chairman of the Eye, Ear, Nose and Throat Section.

Dr. William Zentmayer, by invitation, read a paper upon the "Choice of Operations in the Different Types of Glaucoma," before the Section of Ophthalmology of the Baltimore Medical Society on Nov. 22, 1923.

Dr. Luther C. Peter of Philadelphia, read a paper by invitation at the December meeting of the Lancaster County Medical Association of Pennsylvania upon the "Interpretation of Eye Symptoms, an Absence in General Practice."

The Chicago Ophthalmological Society, at its December meeting, had a symposium on the "Microscopy of the Living Eye." Dr. Blaauw, of Buffalo, read a paper on the "Newer Anatomy of the Lens and the Development of Cataract." Dr. Walter Snyder, of Toledo, discussed the clinical application of the slit lamp and Dr. Sanford Gifford, of Omaha, told of the work being done with the slit lamp in Zurich.

At the meeting of the Section on Ophthalmology of the New York Academy of Medicine, December 17, 1923, papers were read by Dr. Ward A. Holden on "Papilledema, Its Symptoms, Diagnosis and Course"; and by Dr. Charles A. Elsberg, "The Surgical Treatment of Intracranial Disease," with special reference to its influence on papilledema and visual disturbances. A general discussion followed.

MISCELLANEOUS.

The Brooklyn Home for Blind, Crippled and Defective Children has purchased for $100,000 the St. Christopher's Hospital for Babies.

By the will of Charles Elwell Perkins, the Brooklyn Eye and Ear Hospital was left $50,000.

The New York Institute for the Blind will receive $2,000 and the New York Eye and Ear Infirmary $1,000 by the will of Louis T. Lehmeyer.

Thanks to the very efficient laws in Chicago for the prevention of blindness, of 56,724 children born there, not one is blind from preventable causes.

A measure has been approved in St. Louis, permitting cigar stands to be installed in the lobbies of public buildings and operated by blind persons selected by the Commission for the Blind.

The French Ophthalmic Hospital, Hanoi, Tonkin, China, reports from 1917-1920 a large increase in the number of trachoma cases coming for treatment. During that period, 16,625 operations were performed and of these, 13,576 were for trachoma and its complications.

Professor I. Barraquer of Barcelona, much to his regret, finds it is not possible to give his special course as announced in this department in the October issue, vol. 6, p. 870. It is hoped that such a course can be announced subsequently.

The Rotary Club of Panama City has inaugurated a campaign for providing without cost suitable glasses for children who are unable to purchase them. Thru the kindness of a physician, who is a member of the club, and an optical manufacturer, also a member of the club, examinations will be made free of charge and glasses will be furnished at their cost to the club.

Papers which are too long for insertion in one or even two numbers of the British Journal of Ophthalmology are occasionally submitted to the Editorial Committee. The subdivision of such papers is always unsatisfactory and in some cases it would be quite impossible for the journal to allot the necessary space. The Editorial Committee has in several instances been forced to refuse monographs which were worthy of publication owing to this cause. It has, therefore, been arranged, with the sanction of the directors, to issue from time to time Monograph Supplements. These will be published in the same form as the journal, and will be issued at a reduced price to subscribers to the journal. We have pleasure in announcing that the first Monograph Supplement, now ready, is the Gifford Edmunds Prize Essay on "Interstitial Keratitis," by Mr. W. T. Holmes Spicer.

Current Literature

These are the titles of papers bearing on ophthalmology. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in **heavy-faced type.** The abbreviations mean: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is an abstract of the original article. (Bibl.) means bibliography and (Dis.) discussion published with a paper. Under repeated titles are given additional references to papers already noticed. To secure early mention, copies of papers or reprints should be sent to American Journal of Ophthalmology, 217 Imperial Building, Denver, Colorado.

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